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MEDICAL DIAGNOSIS

WITH

SPECIAL REFERENCE TO PRACTICAL MEDICINE

A GUIDE TO THE KNOWLEDGE AND DISCRIMINATION OF DISEASES

BY

J. M. DA COSTA, M.D., LL.D.

PHYSICIAN TO THE PENNSYLVANIA HOSPITAL, ETC.

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NINTH EDITION, REVISED

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EXTRACT FROM PREFACE TO THE FIRST EDITION.

My chief aim in writing this work has been to furnish advanced students and young graduates of medicine with a guide that might be of service to them in their endeavors to discriminate disease. I have sought to offer to those members of the profession who are about to enter on its practical duties a book on Diagnosis of an essentially practical character,—one neither so meagre in detail as to be next to useless when they encounter the manifold and varying features of disease, nor so overladen with unnecessary detail as to be unwieldy and lacking in precise and readily applicable knowledge.

In executing my undertaking, two plans offered themselves: either to describe morbid states in compliance with the usual pathological classification followed in treatises on the Practice of Medicine, or to group them according to their marked symptoms. The former plan would have been far the easier, but the latter seemed to me the more suitable for a volume of this kind; and, although it has involved much labor, and has rendered the task much more difficult of accomplishment, its advantages appeared to me so great that I have adopted it throughout. That this attempt at a purely clinical classification is not perfect, I am fully aware. But, with all its shortcomings, I venture to hope that it will not be devoid of value.

Some of the statements made may appear too absolute, and as not taking sufficient notice of the many exceptions that may arise. But it was impossible to avoid this without lengthy discussion: and even in

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PREFACE TO THE NINTH EDITION.

CONSIDERABLE new matter has been incorporated in this edition, especially in the chapters on the Diseases of the Stomach, on the Blood, and on Fevers, as well as whatever of bacteriological interest has proved of value for diagnostic purposes. Fresh illustrations, too, including some skiagraphs and colored plates, have been added. By a revision of the text, and the rewriting of such portions as now, with our more definite knowledge, admit of greater precision of expression, the additions have not enlarged the book; indeed, the different size of the page and a new type have made it a smaller and more convenient volume. All the fresh matter has been added in accordance with the clinical classification inaugurated when the work was originally written, and which has proved a useful plan. I express with pleasure my indebtedness to Dr. Eshner, Dr. Woodbury, and Dr. Leffmann for valuable aid, as well as to Dr. Leonard for the excellent skiagraphic pictures. The delay in issuing this edition has been caused by the great fire that destroyed the building of the publishing company and, with it, all the copies of the just printed volume. The work is now before the profession in the English, German, Italian, and Russian languages.

1700 WALNUT STREET,
June, 1900.

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MEDICAL DIAGNOSIS.

INTRODUCTION.

GENERAL CONSIDERATIONS.

THE study of any complicated subject leads of necessity to its arrangement into branches. Closely connected as these are, and forming always parts of a whole, they are not only capable of distinct treatment, but frequently become more intelligible as they are so treated. This is made very manifest in investigating disease. The extent of ground covered by the inquiry has rendered it imperative to map it out into various provinces, which, however intimately united, may be with convenience separately surveyed. One comprises the laws and facts common to individual affections; in another is gathered together all relating to their causes; another embraces the consideration of their detection and the full recognition of their nature. It is the purpose of these pages to examine this department somewhat minutely, and especially that portion of it coming within the range of the practitioner of medicine. In so doing it will become apparent how *diagnosis*, for such the distinction of disease is technically called, is partly a science, partly an art; a science, because it comprehensively takes account of general facts, and of principles based on those facts; an art, because it demands a cognizance of the means, and their application to arrive at the desired result.

To consider, then, medical diagnosis in all its bearings, it will be necessary not only to hold up to view the morbid states met with in the examination of the sick, but also to inquire in what manner they may be most readily recognized and explored, and how their differences may be made available in the discrimination of one ailment from another. In a study of this kind, an investigation of symptoms plays unavoidably a prominent part. In truth, the detection of disease is

the product of close observation of symptoms, and of correct deduction from these symptoms,

The first requirement therefore for an accurate diagnosis is to learn to recognize morbid signs. But the art of observation this implies is not easy, and cannot be thoroughly acquired except by practice. No one aspiring to become a skilful observer can trust exclusively to the light reflected from the writings of others; he must carry the torch in his own hands, and himself look into every recess. The knowledge obtained from reading is, however, serviceable in this way: it aids in overcoming one of the main difficulties at first experienced,—to know where to look and what to look for. There are in almost every affection some symptoms which can hardly escape the merest beginner; but also some which do not appear on the surface, and which to find taxes the skill of the experienced physician. And it is especially in this search after hidden signs that medical information as well as cultivated tact is demanded.

Now, to recognize the manifestations of disease, whether they are or are not readily perceptible, we have to employ our eyes and ears, our sense of touch and of smell. Formerly we could go no farther than these senses unassisted would carry us. But science has lent its aid, and furnished means by the help of which we can detect clearly what before we could not detect at all, or of which at best we caught only a glimpse. We now possess instruments of precision by which we ascertain with accuracy the size of organs and their play. With thermometers we tell the degree of heat of various parts of the body. Specific-gravity bottles, and other measures devised for the purpose, inform us of the relative gravity of fluids. The microscope gives at a glance insight into matters which the naked eye fails even to perceive, shows us crystals in secretions, enables us to count the corpuscles in the blood, and to detect minute and disease-causing specific organisms. The laryngoscope demonstrates the appearance and the movements of the organ of speech. The ophthalmoscope informs us of the state of the vessels in the brain. And chemistry is rendering our knowledge of many morbid states amazingly complete. Then the sagacity of comparatively modern times has taught us how a disciplined ear may detect the workings of disease in cavities into which the eye cannot penetrate; and with the marvel of the Röntgen rays we can now see what is going on in the interior of the body. The effect of all these improved methods of study has been to give an immense impetus to clinical research, and to lead to the construction of a solid groundwork, in striking contrast with the looseness of former times. The advance in diag-

nosis thus attained forms, indeed, one of the most pleasing portions of medical history.

When, by means of the aided or unaided senses, the symptoms of the malady have been discovered, the next step towards a diagnosis is a proper appreciation of their significance and of their relation towards one another. Knowledge and, above all, the exercise of the reasoning faculties are now indispensable. The daily habit of investigating disease; a scrutinizing study of the anatomical lesions; chemistry, with its most searching analyses; the microscope, with the wonders it reveals, are all of little use, unless we have been taught the necessity of placing in connection with one another the morbid signs they lay bare, and of considering in individual cases their respective value. Were it otherwise, the science of diagnosis would be simply a matter of memory. It is, however, this very analysis of symptoms and the lengthy process of induction attending it which make medical diagnosis so difficult. Nor is it reasoning on the ascertained facts alone that is required; the premises may be but probabilities; for, in truth, diagnosis deals at times with the logic of probabilities as much as with the logic of patent facts.

Now, we are greatly aided in appreciating morbid signs, and in interpreting them correctly, by already existing knowledge. We look to landmarks which our predecessors have erected, and the gradually accumulated science of semeiology, rightly employed, furnishes the clue to the discovery of the disease. Thus the stores which medicine has laboriously collected during centuries can be used with advantage by all, and exist for the good of all. But besides this knowledge, the laboratory, with its facilities for solving new and obscure clinical problems, is of immense and constantly growing advantage.

But an acquaintance with semeiology is far from being the sole guide to diagnosis, nor does it at once help to a recognition of the malady. There are few symptoms in themselves distinctive; and often a symptom may be due to one of several causes. Semeiology informs us of these different causes; but to find out the precise meaning of the abnormal manifestation in an individual case, we have to draw our inference from all the signs encountered; to compare them with one another; to seek out those that are in the background. We are thus arriving, step by step, at the explanation of the morbid appearances, the starting-point in deduction always being what is known of the affection the presence of which is suspected, and the symptoms of which we are contrasting with those before us. For the conclusion to be valid and exact, it is of course requisite that each part of the testimony have the proper position assigned to it. In reasoning cor-

rectly on symptoms, the same laws apply as in reasoning correctly on any other class of phenomena: the facts have to be sifted and weighed, not merely indiscriminately collected. And while this intellectual act is being performed, much collateral evidence is to be sought before a final judgment is given; especially is it necessary to view the symptoms with constant reference to the age, sex, and habits of the patient, and to the circumstances amid which the disorder develops.

To accomplish all this effectually, the physician has need of much and varied knowledge. He must be master of something more than of the information supplied to him by semeiology. He must be an anatomist to pronounce with certainty on the seat of the malady; a physiologist to appreciate the state of the great centres and the aberration of function. Above all, he must be a pathologist in the full sense of the term: he must understand the antagonism between diseases; the frequency with which they coexist; the influence of remedial agents on them; and be cognizant of their natural history and of the general laws governing them,—for how else can he form an estimate of morbid action while in progress? Then it is desirable that he should be aware of what are their current divisions and classifications. From what has already been represented, it is evident that he must also be a correct reasoner; for even a good observer will, by bad reasoning, arrive at a faulty diagnosis; just as sometimes a bad observer may, by the same process, blunder into the truth. There is, indeed, no end to the extent of knowledge which may be brought to bear in working out a conclusion regarding the character and seat of a malady. The habit of observation once acquired, information of the most varied kind will, by an accurate reasoner, be made tributary to the completeness of the diagnosis. Every fresh acquirement tends to enlarge our powers of insight. Just as in nature, the higher we ascend, the more fully lies the view before us.

Having thus indicated the elements of a thorough diagnosis, we may next inquire in what way this is most easily arrived at when at the bedside. The main facts of the case on which the deductions are to be based are of course first elicited. We lay hold of these main facts, and especially of those that are the most direct signs of the morbid action. They are coupled together, and the inquiry is started as to what organ they indicate as the seat of the malady. This often has been already determined by the very method of the examination; and we therefore proceed at once to investigate the precise nature of the disorder by analyzing the symptoms and the previous history. Sometimes, however, the site of the disease does not admit of being

definitely fixed upon, or we can only in a general manner decide upon the function impaired. Again, as in idiopathic fevers, we may find no signs of local disease,—merely those of a general disturbance. In any of these instances clinical experience steps in to explain the phenomena as far as possible, and to inform us in what affections they occur. It may be only in one; then the desired goal is at once attained. But, as above stated, there are few signs in themselves pathognomonic. It is therefore to be ascertained which one of the disorders is before us that special pathology teaches may yield the symptoms encountered. One of these is taken up. Its symptoms are placed side by side with those present. They accord in some respects, but not in all. Moreover, in searching for some of the phenomena which the supposed malady gives rise to, these are not found. The view is abandoned and another taken up. It agrees in all particulars. The diagnosis is made. Yet, when the diagnosis is thus arrived at, we have still to determine, before it can be considered as complete and can be acted upon, whether or not any other morbid state exists, and to take into account the patient's general condition and his individuality.

To cite a case in illustration. A person consults us for a cough brought on by exposure. He has been ill for four or five days, having been previously in good health. We notice, on examining him, that his breathing is hurried, and that he has fever; the lower portion of one side of the chest is dull on percussion, and the respiration there is wanting; the action and sounds of the heart are normal. The facts point to the lung or its covering as the seat of the malady. We know, furthermore, from the history and the febrile symptoms, that we have to deal with an acute affection. What are the acute pulmonary affections? Acute bronchitis; acute phthisis; acute pleurisy; acute pneumonia. In all occur fever, cough, and disordered breathing. Is it acute pneumonia? No; for, notlıwithstanding there is in this complaint, in addition to the general symptoms mentioned, dulness on percussion, the dulness is associated with a blowing respiration; whereas in the case before us no respiration is heard. Let us look at the sputum, and see if it be tenacious and rusty-colored. It is not; it is thin and frothy. But acute pleurisy may explain all the signs. The patient, too, when questioned, states that he had at the onset a sharp pain in his side; and this, we are aware, takes place in pleurisy. The vocal vibrations, likewise, are noticed to be absent on the affected side of the chest, which, when measured, is found to be enlarged. This corresponds in all points with what happens in pleurisy in the stage of effusion. The disease *is*, therefore, acute pleurisy in the

stage of effusion. We finish the diagnosis by ascertaining the existence or non-existence of other maladies, and by taking note of the severity of the complaint; that it has occurred in a young and robust person of good habits; and that the symptomatic fever is very active.

This process of arriving at an opinion is the simplest. It is one in which the investigation of the case is to some extent carried on while the deductions are being made. And it is astonishing how rapidly it may be performed by habit. The mind works unconsciously, and a decision is, to all appearance, formed intuitively, which surprises the inexperienced by its readiness and precision. This method aims, so far as the symptoms permit, at a direct diagnosis. But, in truth, it is often what is called *differential*,—that is, it takes cognizance of and dwells on the essential signs by which one disease can be discriminated from another resembling it.

Sometimes, instead of attaining the desired result in the manner proposed, we are obliged to judge of the nature of the malady entirely by finding out what it is not. The various diseases capable of producing all, or even some, of the striking symptoms observed, are enumerated. They are one by one considered and set aside, until by this process of pure exclusion the mischief is brought to light. Thus, to use again the example just given, we should have to assign reasons why the disease is neither acute pneumonia, nor bronchitis nor acute phthisis, and in this way determine it to be acute pleurisy. But to prove what a thing is by proving all that it is not is a very tedious process, and we must be quite certain that really *all* morbid states which may give rise to the symptoms encountered are thought of and inquired into; otherwise our conclusion may be fallacious, though reasoned out in the most logical manner. Moreover, our knowledge of many pathological conditions is so imperfect that we are not fully cognizant of, or able at once to discern, the more characteristic signs; nor can the symptoms be taken hold of and arranged in such a way as shall permit us to make nice distinctions without a lengthy and laborious plan of procedure. Owing to these drawbacks, *diagnosis by exclusion* is not, on ordinary occasions, much employed, nor, indeed, is it to be recommended. Yet in difficult and obscure cases, where the accustomed pathway is blocked up, it may enable us to pass by obstacles otherwise insurmountable.

But can we by this or by any other road always reach a certain diagnosis? We cannot, and for several reasons. The patient may deceive us, wilfully or unintentionally. It may be necessary, for the confirmation of the opinion formed, to obtain an accurate history of the case, and circumstances may render this impossible. The dis-

order may be so rare that its symptoms are not understood. There may be several lesions present, the signs of one masking or neutralizing the signs of the other.

The first of the causes mentioned is a source of error difficult to guard against. To escape punishment, to avoid disagreeable duty, to excite compassion, to obtain a compliance with unreasonable wishes, or sometimes from the mere love of deception, symptoms may be stated to exist which do not exist, or may be imitated and artificially produced. Persons who thus feign disease are numerous. They are found in all occupations and in all classes of society. They abound in the army and navy. Hysterical women and hypochondriacs help to swell the list. These, indeed, suffer mostly some inconvenience, but exaggerate it immensely, and, by deceiving themselves, end by deceiving, unless he be on his guard, their physician. On the other hand, disease actually in progress may be carefully concealed from motives of delicacy or from fear of the consequences.

An incorrect diagnosis from want of a proper history does not, on the whole, occur often. Patients are generally very willing to give a full account of themselves and of their distresses. Sometimes, however, the reverse happens. Pain or mental anxiety and sorrow may be wearing the body out while the sufferer obstinately persists in hiding the cause of his waning health. We meet also with individuals so stupid that the most elaborate cross-examination fails to elicit anything like a connected history. Again, we may be unable to do so from the patient having lost the power of speech or being unconscious.

In the rarity of a disease we have a serious drawback to its recognition. This may occasion an error of diagnosis in a twofold manner. The more distinctive symptoms may be so little understood, and the prominent features be so nearly identical with those of a malady with the manifestations of which we are well acquainted, that a conclusion of the presence of the latter forces itself almost immediately on the mind. Or, the disorder may give rise to phenomena wholly unknown, nothing but the autopsy revealing their true meaning. Every physician encounters such cases. It is true that the progress of science and the aggregation of clinical facts are from year to year bringing them into a narrower circle. Yet, are there not still diseases, nay, groups of diseases, that have eluded discovery to the manifold means of research of the present day, as they have to the accumulated experience of the past?

But the most serious obstacle to a precise diagnosis lies in the fact that frequently lesions coexist. Disease is a very complex state, and

when one portion of the economy gets out of order, another is apt to follow. Then a part contiguous to one chronically affected may be attacked with acute disease; or remote sympathetic derangements become very prominent. A thorough examination of the case is the only safeguard against error.

These, then, are the various causes which render a diagnosis uncertain, or wholly unattainable. Let us add to them one that does so temporarily. There are disorders the early manifestations of which are so much alike that it is next to impossible to tell with which of several we have to deal. In fevers this often happens. Here, however, a few days will almost always solve the difficulty. But not so in other diseases. It is only after a much longer period that the appearance or disappearance of a striking symptom, or the greater prominence a hitherto indistinct sign assumes, enables us to reach a decision.

In some such instances, the treatment becomes the touchstone of the diagnosis. Now it may be asked, Does this demonstrate that the diagnosis of a case is not necessary for its treatment? Not at all. It simply proves that we are sometimes obliged to aim at removing symptoms without understanding their source. But it does not prove that if we understood their source we should not be better able to remove the symptoms. The physician who undertakes to relieve disease simply by attempting to allay its symptoms, regardless of their cause, and without understanding their true relation and significance, is groping in the dark. His treatment is vacillating; drug replaces drug; alleviation is taken for a cure; and the experience obtained is utterly untrustworthy. One great advantage, indeed, of attending carefully to diagnosis is, that it enables us to use remedies knowingly and with decision; to appreciate what they are effecting; to abstain from such as must be injurious. There is less needless meddling, more calmness; the treatment rises above the consideration of the moment, and takes into account what is for the patient's ultimate good. But, in basing the management of a disease on its diagnosis, we must never be unmindful how important it is to found that diagnosis on a general survey of all the circumstances; how necessary not to assign prominence to minor points; and how the extent of the affection, the circumstances under which it has occurred, the sympathetic disturbances produced, and the vital state of the patient, belong, rightly considered, quite as much to the diagnosis as the recognition of the precise seat and exact anatomical character of the malady, and are, in truth, frequently its more important part.

CHAPTER I.

THE EXAMINATION OF PATIENTS, SYMPTOMS OF GENERAL IMPORT, AND SOME OF THE INSTRUMENTS EMPLOYED IN THE DIAGNOSIS.

To elicit the facts of a case by a careful examination is, as has been stated, the first requisite for diagnosis. To conduct, however, a clinical inquiry with precision and facility, requires continual practice, and is rendered easier by following some well-digested plan. The advantage of adopting a method is clearly seen, if the attempts of a beginner be watched. He wanders in his search from one part of the body to another, attracted by different symptoms in turn; pointless question succeeds to pointless question; and a conclusion, almost certainly erroneous, is finally jumped at, or an acknowledgment made of inability to arrive at any.

Now, there are several ways which have been proposed to overcome this embarrassment. One of the principal consists in first questioning the patient with regard to his history. His age; his occupation; the diseases from his childhood up; his habits; his constitution; the affections hereditary in his family, are all minutely inquired into. After this the origin and progress of the existing disorder are traced, and the remedies ascertained that have been used against it. The present condition is then explored; each organ or each system being in turn interrogated. The investigation is now regarded as complete; the facts are considered, and the diagnosis, prognosis, and treatment determined. This method of examining is termed the *synthetical* or historical. The *analytical* reverses the order. The present condition is first ascertained, and subsequently the patient's history or *anamnesis*. Both of these courses have something to recommend them, and to both there are objections. The synthetical method is the more purely scientific; but it is too full, and calls for too much labor, to meet the requirements of ordinary professional life. It is much better adapted for recording cases in the pursuit simply of pathological knowledge, and decidedly the best where the history is obscure and the symptoms are ill defined. The plan which I habitually prefer is to take a general survey of the history and of the prominent symptoms, and, having thus obtained some clue to the part most likely to be affected, to explore this with care. For instance: we are

brought to the bedside of a patient for the first time; we inquire how long he has been ill; how that illness began; in what way he is now disturbed,—whether he has pain, or what is the main source of his annoyance. While questioning him, we are scanning his appearance, the position of the body, his movements, his manner of breathing. The hand is applied to the skin: the pulse is felt; the tongue is looked at; the temperature is taken. Partly from this examination and partly from the history, some organ is fixed upon to be specially investigated: say pain in the epigastric region and vomiting are complained of,—our attention is directed to the stomach. We explore this organ, its physical state and its functions. Then we look to the parts that are anatomically or physiologically most nearly related to it, which are, in the case cited, the intestines and the liver. The examination is completed by taking heed of the condition of other portions of the body; by reviewing the history of the case; and by endeavoring to elicit fully such points as bear upon the diagnosis, which the mind, consciously or unconsciously, has begun to frame. Then a balance between the symptoms is struck, the diagnosis is recast, modified, or extended, and the treatment is decided upon.

There is some repetition in this plan, but it is the one which appears practically the most suitable. It has the advantage of bringing together the marked features of a case, and especially those most clearly indicative of the general or vital condition. But whatever scheme is chosen, it should, for us to become proficient in it, be as constantly and closely adhered to as the varying circumstances of disease will permit. Yet to acquire thoroughly the habit of examining with accuracy and care, and also to obtain the full fruits of experience, it is indispensable to keep written records. This, too, should, so far as possible, be done according to a uniform design, since it both prevents us from overlooking important symptoms and enables cases to be more readily compared. I subjoin a schedule that is based on the plan of examination just mentioned.

Date of examination; name; age; color; place of birth; present abode; occupation or social state; in females, whether married or not, number of children, and date of last confinement; how many miscarriages.

HISTORY.

1. *History antecedent to present disease:* Constitution and general health—Hereditary predisposition—Previous diseases or injuries or taints—Habits and mode of life; hygienic influences to which exposed, etc.

2. *History of present disease:* Its supposed exciting cause—Exposure to contagion—Date of seizure—Mode of invasion; subsequent symptoms in order of succession—Previous treatment.

PRESENT CONDITION OF PATIENT.

Height and weight.

1. *General symptoms:*

Position { in bed—mode of lying; / out of bed—movements; gait and station;

Aspect { of body; / of countenance;

Skin;

Pulse;

Temperature;

Respiration—as to frequency, character, etc.;

Tongue;

General state of digestion { appetite; / thirst; / condition of bowels;

General state of urinary secretion and urinary analysis;

Sensations of patient: pain, etc.

2. *Examination of special regions, parts, and functions*, beginning with the one presumably the most affected, and embracing, whenever practicable, microscopical examination of the blood and bacteriological studies.

DIAGNOSIS.

TREATMENT.

REMARKS.

The history is here placed first; then the symptoms of general import, such as those furnished by the pulse, the tongue, and the temperature, are made to precede the examination of special regions. These general symptoms are of great value in the recognition of disease, and of yet greater value in determining its treatment. They are more than the mere physical signs of textural affections; they indicate vital conditions, and partly from their importance, and partly from their not being linked to disease of any organ in particular, they demand a separate and detailed consideration.

Position of the Body.—By noting whether the patient is in bed or out of bed,—how he lies, or how he walks,—a general idea may be formed as to the acuteness of an attack, the impairment of strength

it has produced, and sometimes even as to its nature. Let a person who has been actively attending to his usual occupation be suddenly confined to his bed, and the inference that the disease is an acute and a severe one will be commonly correct; certainly so, if no mishap to the organs of locomotion have necessitated a resort to the recumbent position. When the patient lies for a long time on his back, it is generally from exhaustion, or from paralysis, or it is owing to the pain which pressure or motion of any kind occasions. Such is the cause of the dorsal decubitus in peritonitis, and in rheumatism. Lying steadily on the back with a disposition to slip down in bed is a form of dorsal decubitus witnessed in low fevers. Lying fixedly upon one side may, as a rule, be looked upon as an indication that the action of the lung of this side is impeded, and that the respiration has to be carried on with the other. The patient may be confined to bed, yet unable to lie down in it, on account of the distress in breathing to which the recumbent posture gives rise: he leans forward, or sits erect. This necessity of breathing in the upright position, or "orthopnœa," is a form of dyspnœa encountered especially in diseases of the heart, or where fluid is effused into the air-cells or into both pleural cavities.

If a person is able to be about, his posture and movements become important manifestations of his condition. The young and the strong walk erectly, quickly, and firmly; the aged and the weak, stoopingly, slowly, and with difficulty. In diseases of the spine the body is bent; so, too, in affections of the larger joints of the lower extremities.

When, after a fever or any other prostrating malady, the patient leaves his bed, he totters, moves slowly, and is soon obliged to rest: returning strength brings with it a quicker and steadier gait. In some diseases of the brain the movements are very uncertain; in one-sided palsy the affected side lags, or its motions, if it can be moved at all, are laborious. Excessive and uncontrollable movements are observed in mania and in chorea; trembling motions in states of extreme debility, in shaking palsies, and in the delirium of drunkards; irregular motions and positions chiefly in hysteria.

The *gait* is always to be closely studied. We find it of special significance in affections of the nervous system and of the muscles. It is very erratic, from side to side, in locomotor ataxia, and there is almost total inability to walk in the dark. In paralysis agitans the tremors are associated with a festinating gait, each step becoming more rapid than the last, and a fall is only averted by seeking support. In spastic paraplegia the legs drag behind; in walking each leg is rigidly brought forward, the toes having a tendency to catch the

ground. In pseudo-hypertrophic paralysis occurs a peculiar oscillating or waddling gait, from weakness of the extensors of the knee and hips; there is also much difficulty in rising from the ground. In Thomsen's disease it may also be for some time impossible to rise from the floor, and the gait is at first impeded by tonic spasm of the muscles.

Station, or the power of preserving an erect position while standing, is often as characteristic as the gait. It should be noted while the eyes are open, while they are shut, and while the feet are placed alongside each other with the heels and toes touching. Under both the latter circumstances the station is always less certain and the swaying of the body more marked. Tested with an instrument invented by Weir Mitchell,[1] Hinsdale[2] found in the normal man and woman the average sway, while the heels and toes were touching, to be about an inch in the forward and backward line, and three-quarters of an inch laterally. Children sway to a greater extent than adults. Closing the eyes increases the sway about fifty per cent. In locomotor ataxia station is much disturbed and the sway greatly increased; so it is in disease of the middle lobe of the cerebellum. In the attacks of aural vertigo all power of standing may be lost.

General Aspect—Expression of Countenance.—A bulky aspect of the whole body is the result of corpulency or of universal anasarca. In the exanthemata, too, a general tumefaction may take place. A partial increase or a swelling arises from the local extravasation of fluid or air into the cellular tissues. If air, the tissues crepitate under the finger; if fluid, the skin pits under pressure. A swelling may also proceed from an inflammatory thickening or from a tumor or any morbid growth.

A diminution in bulk is a more frequent symptom than an augmentation. It may occur rapidly, as in Asiatic cholera. More generally the wasting is gradual, and is an indication of defective nutrition. It happens in the course of protracted fevers, and in most chronic diseases, especially in those attended with constant discharges.

Emaciation is most readily recognized in the face. But it is not the only striking alteration observable in the countenance when health has failed. There may be pallor, sallowness, a livid hue of the lips, a puffy appearance of the eyelids, a flush on the cheeks. Now, these changes in the features, added to the expression which pain or special trains of thought produce, make up the physiognomy of disease so pregnant with meaning.

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[1] Amer. Journ. Med. Sci., 1887.

[2] Ibid., April, 1887.

Among the countenances most frequently met with is that of apathy and stupor. The eye is dull and listless; the face pale or flushed. This look is common in fevers of a low type, and is often combined with blackish accumulations on the lips, gums, and teeth.

Unnatural fulness and congestion of the features are sometimes observed in enlargements of the heart, and oftener still in habitual drunkards. The same aspect is seen in apoplexy and in typhus fever. Local congestions on the cheeks and nose are met with in obstructive diseases of the liver, especially in cirrhosis, and in the endarteritis of old persons. A pinched expression is found when there is intense anxiety or pain, or a wasting malady attended with constant suffering. It is specially observed in acute peritoneal inflammation. When very marked, and accompanied by change of hue, it is the face which Hippocrates has so graphically described. In the great master's own words, "a sharp nose, hollow eyes, collapsed temples; the ears cold, contracted, and their lobes turned out; the skin about the forehead being rough, distended, and parched; the color of the whole face being green, black, livid, or lead-colored." This is the physiognomy of approaching death, and generally its speedy forerunner, except in those cases in which the expression proceeds from want of food, from protracted vigils, or from excessive intestinal discharges.

The face of shock, with its great pallor, its anxious or frightened look, and its fixed or oscillating eye, often with a contracting pupil, is a face seen after severe injuries, and as such familiar to the surgeon. But in many of its main traits it may be also met with in diseases that make a sudden and overwhelming impression on the nervous system; for instance, it is at times encountered in cerebro-spinal fever and in cholera.

Besides these lineaments, which may be said to be common to several diseases, we read frequently in the countenance the signs of special disorders. A dusky flush on the face, if associated with rapid breathing, is almost a certain indication of inflammation of the lung. Puffiness of the eyelids in a pallid person is most apt to be expressive of Bright's disease. A bluish color of the lips shows plainly that the venous circulation is interfered with, or that the blood is but imperfectly aërated. The cyanosis is also recognized in the blueness of the nails and the duskiness of the whole surface. Then there is the chronic pallor of the anæmias with the pearly eye and the yellowish tinge of the pallor in chlorosis; the straw-colored anæmic hue of malignant disease; or we note the jaundiced, melancholy look of an hepatic affection; the downcast expression and mobility of the features in hysteria; the thickened upper lip, delicate skin, and fair

complexion of scrofula; the sallow countenance and peculiar notched teeth that indicate inherited syphilis; the bronzed skin of suprarenal disease; the puffy, vacant face of myxœdema; and the various traits which tend to mark not only the special diathesis, but also the peculiar temperament, with the morbid tendencies that belong to it.

Skin.—By the state of the skin we can, to a great extent, judge of the activity of the circulation and of the character of the blood. Moreover, it is a fair index of the secretions, and of the condition of the system at large. When, after pressure on the skin, the blood returns slowly to the surface, it denotes a sluggish capillary circulation; when rapidly, an active one. Coldness of the surface indicates a weakened capillary circulation, and is met with at the invasion of acute diseases, and when the nervous power is greatly depressed. If the heat of surface succeed a cold skin, we know that reaction has taken place, that the circulation has again become active. Protracted coldness, whether attended with dryness or with clamminess, is of evil augury; it implies seriously diminished vital force.

The cutaneous covering is pale whenever the blood is poor and watery. Black spots may be seen, due to extravasation. Ofttimes the surface is overspread with eruptions, some of which bear a close relation to disorders of internal organs, while others are connected with febrile or general maladies; and others, again, are owing to a disease of the texture itself.

Tension of the skin is met with in acute affections accompanied by active excitement. In wasting and prostrating ailments, on the other hand, the skin feels very relaxed and soft; and in those producing rapid emaciation, it is inelastic and lies in folds.

Pulse.—The pulse enlightens us on the action of the heart, and on the state of the artery itself and of the blood. In a healthy adult a beat of some resistance is felt, recurring from sixty-five to seventy-five times in a minute. It becomes slower with advancing years, though it may rise in the very aged. The pulse of infancy is from one hundred and ten to one hundred and twenty; that of a child three years old, from ninety to ninety-five. Warmth quickens the pulse; so do rapid breathing, forced expiration, and the process of digestion. In the recumbent position and during sleep it falls. For purposes of comparison, the pulse should be, so far as possible, taken under similar conditions.

At the bedside we study in the pulse its frequency, its rhythm, its volume and strength, and its resistance.

Increased *frequency* of the pulse denotes increased frequency of the heart's action, and arises from any cause that excites the heart.

Hence exercise, rapid breathing, mental emotion, or restlessness will occasion the number of beats to exceed the average of health as readily as fevers or acute inflammatory diseases. In great debility, too, the pulse rises ; and the more depressed the vital condition, the higher the pulse becomes. In exophthalmic goitre the pulse is generally very frequent, and rapid heart action may show itself without any other obviously abnormal state, as in tachycardia, a disorder in which the pulse may considerably exceed two hundred beats in the minute. Under the influence of suggestion the cardiac action may be made very much more rapid or slower.[1] As a sequel of influenza there is often very rapid heart action. The heart may thus quicken from so many and such varied causes, acting temporarily or permanently, that increased frequency of pulse, taken by itself, has no significant diagnostic meaning.

A *slow* pulse, too, happens in many different states,—in cold, in exposure to wet, in icterus, in protracted convalescence from acute disease. It is also produced by an intense and prostrating shock, or is found coexisting with pressure on the brain, with melancholia, with atheroma, with fatty heart. A permanently slow pulse is also met with in irritative lesions of the cerebral centres, among them in spherical or pediculated thrombi, in altered state of the circulation in the medulla, and in injuries to the pneumogastric. It is not unusual in instances of very slow pulse, or brachycardia, to observe two or three abortive beats succeeding a strong beat. In some persons the pulse is naturally very slow.

The *rhythm* of the pulse is often perverted. Instead of the beats following one another in regular succession, they are unequal, or one or two intermit. An irregular pulse occurs from digestive disorder, from gout, from lithæmia, from the excessive use of tobacco, tea or coffee, or from nervous exhaustion ; it is less frequently the indication of a cerebral or cardiac lesion. It is sometimes a difficult beat to count ; and we must be careful not to regard at once a pulse as irregular because it appears to intermit. The seeming irregularity may be caused by the fingers slipping from the artery, which they are very apt to do after they have been on the vessel for some time.

Where every other beat is uneven in size, thus showing a beat of greater, followed regularly by one of lesser, altitude, though the rhythm may be regular, we have the *pulsus alternans*. Where a beat is dropped,—in other words, where the heart-beat is not transmitted to the artery with sufficient force to be felt,—it is designated as an

[1] Sgobbo-Nuovo Rivista, 1, 1892.

abortive beat. Two imperfect or abortive beats occurring in rapid succession, and followed by a long pause and generally by a distinct beat, form a linked beat.

The *volume* and *strength* of the pulse are of much more importance than either its rhythm or its frequency. Volume and strength are often associated, and are much alike; but they are not identical. When the beat of the artery is large, we call it a *full* pulse. This is owing to the distention of the vessel with blood,—its complete expansion with every beat of the heart. A full pulse is, therefore, the pulse of plethora; the pulse of the young and robust in health, or in inflammatory diseases; the pulse in the early stages of fevers, or in obstruction of the capillaries. It is usually a pulse of power, just as its opposite, a *small* pulse, is usually the pulse of debility. Yet a full pulse may be produced by the distention of an artery which has lost its tone, and which the finger easily compresses. Such a pulse, the "gaseous pulse," a pulse really of low tension, denotes exhaustion, and proves that a full pulse and a strong pulse are not always synonymous. Into the idea of *strength* something more than mere fulness enters. A strong pulse is a pulse heightened in all its natural characters. It has more fulness, but, in addition, more impulse, and less compressibility, than an ordinary pulse. A strong pulse, therefore, indicates activity of the contraction of the heart, and a normal, perhaps increased, tonicity of the arterial coats. It is found in active inflammations; also in hypertrophy of the heart. Its opposite, a weak pulse, betokens want of force, often want of healthy blood. It is generally small as well as weak. Yet as the full pulse is not always strong, neither is the small pulse always weak. The small choked pulse of peritoneal inflammation may be fine and wiry, but it is not a weak pulse. We also find a small pulse of high tension in mitral stenosis and in contracted kidney.

The *resistance* or tension of the pulse is another valuable guide. A *hard*, tense pulse denotes increased contractility of the arteries, and generally high-wrought power. It tells us that the blood is being driven with force along the arterial system. But it also tells us that the irritation has implicated the coats of the arteries themselves, or that there is obstruction in the capillaries. A tense pulse is met with in active, violent inflammations, and sometimes, though not often, in states of extreme and continued excitement without inflammation. It is almost needless to add that changes in the coats of the arteries may also be a cause of a hard and resistant beat, the common cause of the increased tension in elderly people. Where no local alterations are present, and where no acute symptoms explain the sym-

pathetic disturbance of the heart and arterial system, the high arterial tension will be commonly found associated with hypertrophy of the left ventricle, with interstitial nephritis, with disease of the suprarenal capsule, with gout or lithæmia, or with septicæmia.

The opposite of the hard pulse is the *soft* or compressible pulse. This implies deficient impulsion, and loss of tone in the vessel: it is the pulse of low fevers, of debility, of cardiac weakness. But it is also, when following a tense state of the artery, the pulse which denotes returning health, and danger passed.

When the pulse is of low tension, and at the same time frequent, it may show double beats with each contraction of the heart. This dicrotic pulse is most often met with in fevers of a low form and preceding or during the continuance of hemorrhages. The rebound is chiefly due to the oscillation of the column of blood in the arteries, and is very much influenced by their elasticity. With lowered tension and increased elasticity of the tubes, dicrotism becomes obvious, especially with a rapid circulation. In old persons, in whom the coats of the arteries are inelastic, dicrotism is but feebly marked.

Such are the meanings attached to the various characters of the pulse. Yet they do not often present themselves thus isolated. The following are usually combined, and bear this explanation:

A hard, full, frequent impulse occurs in active inflammations, and in most of the acute diseases of robust persons.

A hard pulse, full or small, bounding or not, if unconnected with acute symptoms, leads to the suspicion of cardiac or of renal disease, or of an affection of the artery itself.

A tense, contracted, and frequent pulse is met with in a large group of inflammations below the diaphragm, as in enteritis, peritonitis, gastritis.

A frequent pulse, full or small, but not tense, is the pulse of most irritative fevers, and with marked low tension is also apt to be dicrotic.

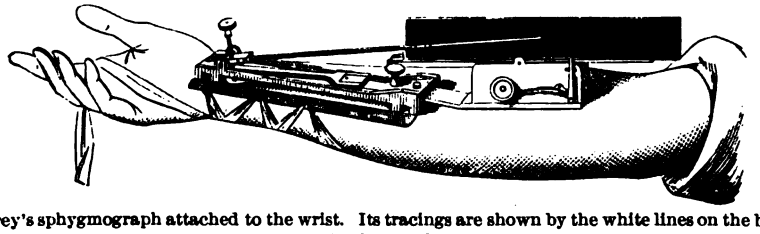
A very frequent pulse, but very feeble and compressible, is the pulse of marked debility, of prostration, of collapse.

A pulse frequent and irregular in its rhythm is produced for the most part by perverted movements of excitation with cardiac disorders, by fulness, by hæmatinæmia, or by disease either of the heart or of the brain.

To recognise readily the shades of difference and to record the movements of the pulse, instruments have been sought. The best of these is the *sphygmograph* invented by Marey (Fig. 1). Slight irregularities that would escape the finger are, through its aid, exercised

with facility, and we know at once in how far these irregularities belong to one beat or to a succession of beats. Double beats, too, not appreciable to the hand, are easily detected. Indeed, the sphygmograph proves the phenomenon of dicrotism to exist in almost every person. The rebound may occur during the systole or the diastole of the vessel; and instead of one, there may be four or five of the secondary pulsations.

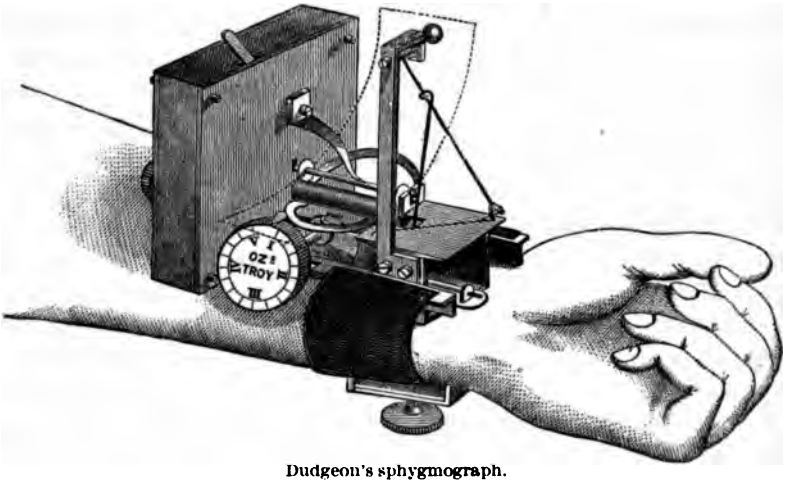
FIG. 1.



Marey's sphygmograph attached to the wrist. Its tracings are shown by the white lines on the black background.

The mode of adjusting the instrument, and of proportioning the pressure of the spring, has something to do with the kind of delineation obtained; and to secure greater accuracy, a number of modifications have been made, chiefly with the view of registering the amount of pressure. The sphygmograph of Dudgeon (Fig. 2) is simple and

FIG. 2.



Dudgeon's sphygmograph.

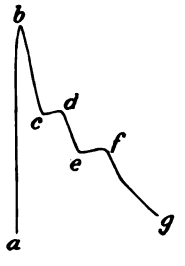
much employed. The system of levers is the same as in Marey's, but the slip of paper moves in a different direction.

To show the tracing distinctly, smoked glass or mica, or paper smoked over a lamp or by burning camphor, is much used; and the

tracing may be preserved by dipping it in an alcoholic solution of shellac or of benzoin, or of a varnish of benzoin and methylated spirit, in the proportion of one to six. On every tracing the amount of pressure employed should be noted. Manifold have been the suggestions to obtain the steadiest application of the instrument to the forearm and the greatest development of the tracing. Lorain[1] has proved that raising the arm to a vertical position gives a much more ample trace; and Richardson[2] shows that with the body in the horizontal line, the dicrotic wave becomes more prominent.

When we apply the sphygmograph for clinical purposes, we study in its tracing the line of ascent, the summit, and the line of descent. Each pulsation is composed of these three parts. The *line of ascent*, the upstroke, tells us the manner in which the blood enters the vessels. The more rapid the flow, and the more quickly the artery distends, the more strictly vertical the line. The force, too, is indicated by this line, or rather by its height: hence when the muscles of the heart contract powerfully, either from enlargement or from overaction, the line is both vertical and high. Yet the strength of the ventricular contraction is far from being the only cause influencing the amplitude of the tracing. Indeed, as we may note in old persons, a large volume of the artery gives considerable height to the lines of ascent; so does a long interval between the pulsations, or the obstruction of the vessel below the point where the observation is made. Low tension in the arteries or in the capillaries has the same effect; whereas when the passage in the ultimate ramification of the vascular system is difficult, the lever descends slowly by a convex line, and is soon again raised by the next pulsation. When the contraction of the heart is feeble, the line of ascent is not vertical or high, but oblique and short. In aneurisms of the thoracic aorta—indeed, in an aneurism interposing anywhere between the heart and the radial artery—an oblique and short upstroke is also met with.

FIG. 3.



SPHYGMOGRAM ENLARGED.—*a*, *b*, upstroke, or line of ascent; *a*, *b*, *c*, percussion-wave; *c*, *d*, *e*, tidal, or predicrotic wave; *d*, *e*, *f*, aortic notch; *c*, *f*, *g*, dicrotic wave; *f*, *g*, diastolic period.

The line joining the summit of a series of pulsations, or the maxima of tension, is generally a straight line; a similar imaginary

[1] The Asclepiad, 1886.

[2] Le Pouls, Paris, 1870.

line connecting the bases, or the minima, is apt to run parallel to it; but irregularity of pulsation leads to irregular lines, and the lower line may be irregular while the upper is straight. Irregularity of the base line is seen in marked dyspnœa.

The *summit* of the pulsation informs us of the time during which the entrance of blood balances the onward flow. A pointed, distinct summit-wave belongs to vigorous contraction of the heart-muscle. The summit may be a horizontal line of some length. This broadening of the apex happens in high and prolonged arterial tension, such as from the slow contraction of a strong heart, fulness of the vessels, or obstruction in the capillaries; an extended plateau is also met with in induration or ossification of the arteries.

In some instances we find a little hooked point preceding the usually transverse mark of the summit. This occurs by the rapid movement of the lever, and is generally a sign of regurgitation co-existing with obstruction at the aortic valves. In aortic narrowing of marked degree the summit-wave is indistinct or absent; the line of ascent is oblique and gradual, and may show a break near the summit.

The *line of descent* is sometimes purely oblique, and the more rapidly the pressure is lessened in the arterial system, the more oblique is the line. It often shows a series of undulations. The first of these waves is called the tidal wave; it is still part of the systole and onward flow of the blood; the decided subsequent wave is specially called the dicrotic or great secondary wave. The closure of the aortic valves with the second sound of the heart happens just before the dicrotic wave; the exact time is marked by the aortic notch; the dicrotic wave represents the diastole of the heart. The tidal wave is large, but the dicrotism badly marked, in atheroma. In high arterial tension the dicrotic wave is also ill pronounced, and the line of descent is very gradual. In mitral narrowing, the line of descent is long, but is broken by small pulsations.

The sphygmograph requires care and practice in its use, and, on the whole, it is of much more avail in investigations on the exact action of medicines—where, indeed, it is of great value—than in aiding us materially in questions of diagnosis or in decisions on treatment. At all events, I do not think that it supersedes the older and more usual means of research. Perhaps records of pulse-traces in which the amount of pressure has been carefully noted will enable us to judge more accurately than we can now of the state of the cardiac muscles in disease.

An instrument aiming at even greater accuracy than the ordinary

sphygmograph is the *sphygmochronograph.*[1] It is similar in its construction to the sphygmograph of Dudgeon, but it enables us to measure the curves of the tracings, and to ascertain the exact time of each part.

Normally *capillaries* do not pulsate. We judge of their dilatation by the flush, of their contraction by pallor. But in certain pathological conditions they beat, as may be observed in the capillary flush. We may note the *capillary pulsation* in instances of chlorosis and of aortic regurgitation. The capillary flush has generally to be brought about artificially by pressure on the skin, the nails, or the lips. We can then perceive the pink changing in color with each pulsation, or disappearing after it. The most marked changes are observable at the periphery of the pink patch. In those rare instances in which the capillary pulse is regurgitant and of venous origin, as in tricuspid regurgitation, we find venous pulsation everywhere, and the capillary pulsation precedes the radial heat.

Temperature of the Body.—The thermometry of disease is indispensable. The thermometer used for clinical purposes should be very sensitive, and requires to be from time to time compared with a standard one, and verified; it should be self-registering. The detached part, or the index, is set by bringing it down below the lines of the scale by a rapid swing of the arm; a magnifying front allows the degrees to be easily read. Very delicate but fragile thermometers, registering in a minute or less, have of late come into use. Metallic thermometers are neither so cleanly nor so trustworthy as those made of glass.

As surface thermometers for localized thermometry various instruments have been suggested. I habitually employ one which has the mercury in a fine coil at the expanded extremity, and which is self-registering. We should first obtain the heat of a corresponding well part, and then leave the bulb for five minutes on the suspected abnormal structure. Better still is it to apply two instruments at the same time; one on the sound, the other on the unsound side. In all observations the heat of the body, as ascertained in the axilla, should equally be noted.

The surface temperature is, as a rule, lower by upward of one or by several degrees than the general temperature. We find it so on the chest, on the abdomen, and on the head. The temperature, too, is not on corresponding sides entirely the same, at least not on the head. There is almost always a slight inequality in the temperature of the

[1] Jaquet, Zeitschrift für Biologie, 1891; and Mühll, Deutsch. Arch. f. klin. Med. 1892, xlix.

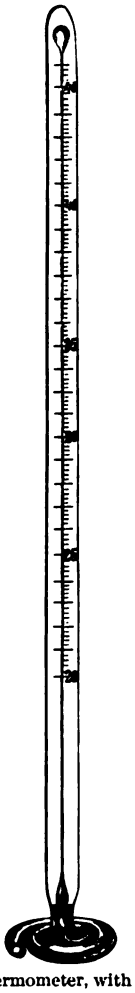
FIG. 4.

Self-Registering Thermometer, showing the index marking 99° F. shortly after an observation.

FIG. 5.

Seguin's Surface Thermometer, modified to be self-registering.

FIG. 6.



Surface Thermometer, with coil at extremity. It may be, if necessary, kept in place by a thin elastic band.

two sides of the *head;* Gray[1] demonstrates that when at rest the temperature of the left hemisphere is the higher, which accords with Broca's statement. And the observations of Amidon[2] have shown that excessive use of a group of muscles may generate heat, in the cortical centres presiding over them, sufficient to manifest itself to surface thermometers placed on the scalp; emotional and intellectual activity Lombard has proved will do the same. The mean temperature of a healthy man's head is fixed by Maragliano and Seppili, as the result of many observations, at 36.13° C. (97.03° F.) for the left side of the head, and 36.08° C. (96.9° F.) for the right.[3] These temperatures are higher than those given by Broca and Gray. Broca places the frontal region on the left side of the head at 35.43° C. (95.79° F.), on the right at 35.22° C. (95.39° F.). The parietal region on the right side is fixed by Broca at 92.8°; by Gray at 93.6° on the right, and 94.4° on the left; the vertical by Gray at 91.7°, and the occipital at 91.9°; the whole side of the head by Broca at about 93°; the entire head at places remote from these points at 93.5° by Gray.[4] In furious mania a temperature of 36.9° C. has been observed, and a rise of temperature has also been noted over brain tumors, cerebral abscesses, and tubercular inflammation.[5] But, on the whole, cerebral thermometry has not proved itself of much value.

As regards the abdomen, Peter[6] places the normal mean of the parietes at 35.5° C. (95.9° F.), and the same observer records the normal temperature for the chest-walls at about 36° C. (96.8° F.). Certain diseases change the temperature locally. Thus, in neuralgia the heat near the painful points may be markedly raised. So, too, is it sometimes in some parts of the surface in hysterical women. In hemiplegia the paralyzed limb may show a higher temperature than the sound one; and over spots where there is inflammation or where decided tissue-change is going on there is a rise in local temperature. Weir Mitchell[7] has called attention to the manner in which posture affects surface temperature. It is, for instance, less by 0.4° C. to 1° C. on the dorsum or sole of the foot when standing than when lying down.

But to return to general thermometry. The clinical thermometer

[1] Chicago Journal of Mental and Nervous Diseases, 1879.

[2] New York Archives of Medicine, April, 1880.

[3] Translated in Alienist and Neurologist, St. Louis, Jan. 1880.

[4] New York Archives of Medicine, 1879, vol. ii.

[5] Eskridge, Transactions of the College of Physicians of Philadelphia, 1883.

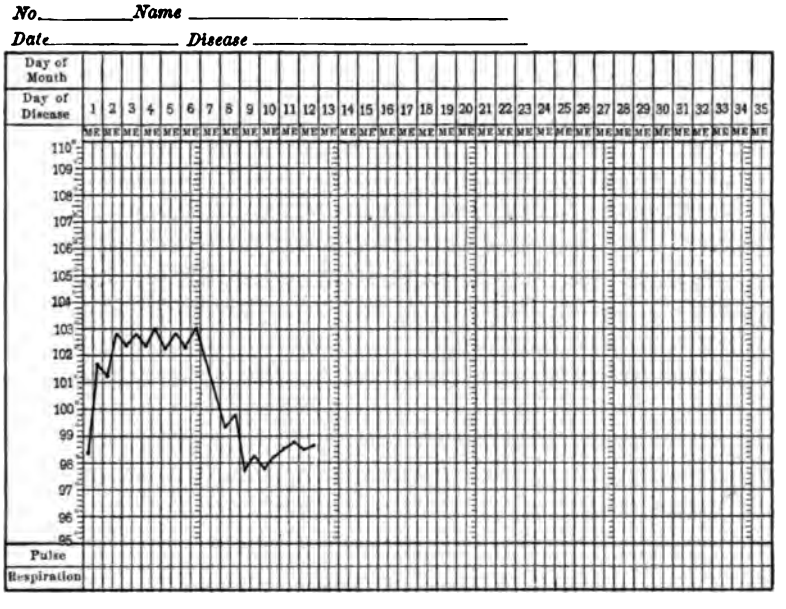
[6] Communication to the Académie de Médecine, quoted in Medical Times and Gazette, Dec. 1879.

[7] Medical News, Jan. 1894.

may be put under the tongue or in the rectum; but the most suitable site in adults is the axilla. The bulb is pressed into the armpit and kept in close contact with the skin for five minutes, except when the delicate minute thermometers are employed. The thermometer may be conveniently introduced just below the skin covering the edge of the pectoralis major muscle; and, to insure exactness, the axilla should be kept well covered. In using the thermometer in the mouth we must be careful that it be not used soon after anything hot or cold has been taken. The effect of heat in the mouth is more prolonged than of cold.[1]

In all cases of importance, not less than two observations should be made daily, and, so far as possible, every day at the same hour. Between seven and nine o'clock in the morning, and about seven o'clock, or somewhat earlier, in the evening, are regarded as the most

FIG. 7.



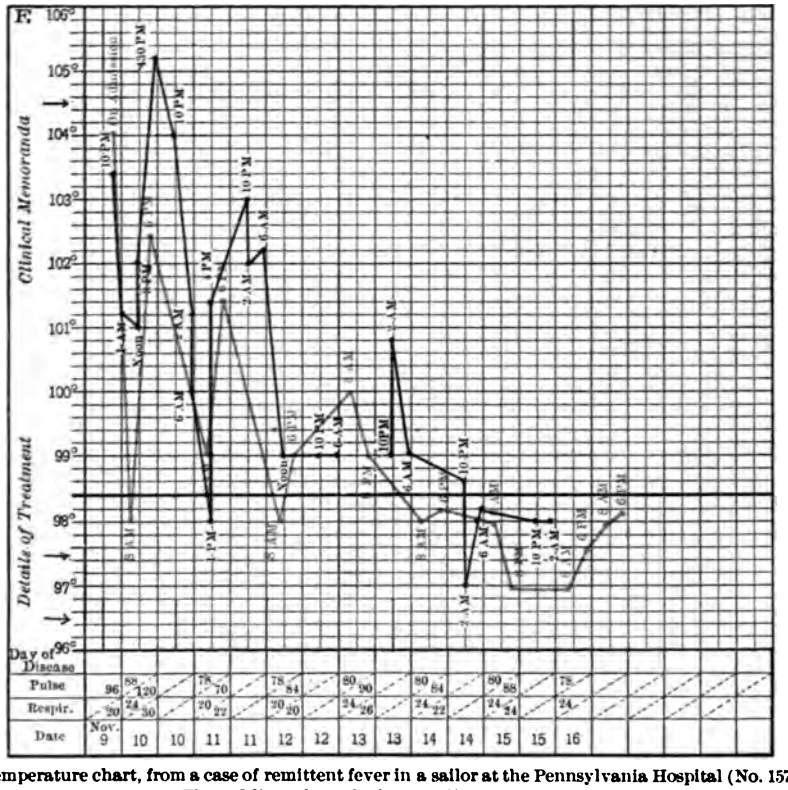
Temperature chart in simple continued fever. The *initial* stage, the stage in which the temperature rises to its height, is here attained on the second day; the *fastigium*, the stage at which it remains, with slight fluctuations, at its height, lasts until the sixth day; the *defervescence*, the stage when the fever-heat falls, is rapid, by crisis, and a subnormal point is, for a time attained.

appropriate periods. If only a single observation be taken, it is best done in the late afternoon or in the evening. In every record of the temperature the pulse and the respirations must also be noted. For the purpose of record clinical charts should be always used. Fig. 7

[1] Lazarus-Barlow, Lancet, Oct. 1895.

is a simple model. It can be arranged for thirty days or upward; by striking out the morning and evening marks, as many spaces as needed can be devoted to a case; or by using a marking in red ink in addition to the black line in which the morning and evening temperature is recorded, as near as may be always at the same hour, the supplementary temperature can easily be traced (Fig. 8). Where we wish also to

FIG. 8.



Temperature chart, from a case of remittent fever in a sailor at the Pennsylvania Hospital (No. 1570). The red lines show the intermediary temperatures.

show the pulse and the respiration graphically, the chart of Crozer Griffith is excellent. In discussing pneumonia, farther on, one of these charts is shown.

In temperate climates the average heat of the body, as measured in the axilla, is estimated at 37° C. (98.6° F.);[1] that of freshly voided

[1] It may be useful, for the sake of comparison, to recall the fact that one degree of Fahrenheit is equal to five-ninths of a degree of the Centigrade thermometer, and four-ninths of a degree of Réaumur; and also that the freezing-point of the

urine is about the same. This, at least, is the case in the axilla; in the rectum it is not quite one degree higher, and is very steady; in the mouth it is somewhat lower. In the groin, where, in children, it may be most convenient to take it, the temperature is apt to be lower than in the axilla.

The body temperature rises with the temperature of the air, and fluctuates slightly during the day, being in temperate climates, according to the most trustworthy observers, lowest between two and eight in the morning, and highest late in the afternoon. It is heightened by exercise and reduced by sustained mental exertion, and changes even when we are at rest. But, as a rule, with the exception of very active exercise, no cause save disease induces a variation of much more than one degree; even in the extreme heat of tropical climates the animal heat does not surpass 99.5°. Thus a temperature above this, or more than a degree below the average stated, when persistent, indicates some morbid action in the economy. At all events, it does so in adults; in very aged persons a temperature of 97° may still be normal; while, on the other hand, the range may be as high as in infants.

In children, in whom the temperature, as a rule, is somewhat higher than in adults, the daily range is much greater. It falls rapidly in the evening, and is very much influenced by food and by crying. In the new-born it is about 99.8° to 100.4° in the rectum. It falls from early infancy to puberty. The rectal temperature of young children ranges between 99° and 99.7°; under six years of age the mean is 99.4°. The maximum is attained in the afternoon. During the first three or four months of life the temperature, Henoch asserts, has, from slight causes of faulty nutrition, a marked tendency to go below the normal. A further point, too, to be taken into account in those of all ages is, that the temperature is somewhat influenced by food and stimulants and by prolonged application of the thermometer. And these are the elements which make deductions from single observations or comparatively slight changes untrustworthy. In high altitudes, as Keating[1] has observed, there is a tendency to hyperpyrexia.

In ordinary cases the pulse and temperature rise synchronously,

first is placed at 32°; that of the others at zero. To convert Centigrade into Fahrenheit, we multiply by 9 and divide by 5; to convert Réaumur, we multiply by 9 and divide by 4; and when above zero, in either case, add 32. To convert Fahrenheit above zero into Centigrade, we subtract 32, multiply by 5, and divide by 9.

[1] International Medical Magazine, Dec. 1892.

and every degree above 98° F. corresponds with an increase of ten beats of the pulse. The fever temperature ranges from 100° to 106°. When it exceeds this, the patient may be looked upon as in great danger, except the rise be due to malarial fever. Under these circumstances it is rapid, occurring in a person who but a few hours before was healthy. In typhoid fever a temperature of 105° is a proof of grave disease. In some severe cases of yellow fever the heat in the armpit has been noted as 108°.[1] In pneumonia a temperature above 104° is a symptom of a very serious seizure; so, too, is it in acute rheumatism a symptom either of danger or of some complication. Stability of temperature from morning to evening is a good sign; the temperature remaining the same from evening till morning is a sign that the patient is getting worse. In convalescence the temperature declines until it attains its norm, or even falls somewhat below this. If after the defervescence the thermometer again indicate a decided rise, it shows a return of the malady, or the supervention of some complication or new disorder; and the persistence of even a slight degree of abnormal heat after apparent convalescence is a sign of imperfect recovery, or of the existence of some lingering secondary complaint. Further, in cases of low fevers, the skin, particularly of the hands and feet, may feel cool at the same time that the instrument in the axilla marks 104°.

Specific forms of febrile diseases have their characteristic variations of temperature. In measles, for instance, the temperature rises towards the breaking out of the rash, reaches its height with the period of eruption, and in the twenty-four hours succeeding it falls rapidly. In scarlet fever the thermometer marks 105°, or upward, at the beginning, and the heat only gradually subsides. Typhoid fever has its characteristic record; so have the malarial fevers theirs. The temperature of tetanus rises to great heights before death.

A temperature about 107° is almost certain to be the forerunner of a fatal issue. But recovery may take place. In a case of cerebral rheumatism under my charge[2] the thermometer marked 110° in the axilla, yet the patient got well. In an instance of injury to the spine after a fall, reported by Teale,[3] the young lady lived though the temperature reached above 122° and ranged for days between 112° and 114°. A remarkable case has also been reported of hysteria and intercostal neuralgia, in which in one axilla the temperature

[1] Wragg, Charleston Medical Journal, vol. x.

[2] See Amer. Journ. Med. Sci., Jan. 1875.

[3] Transact. Clinical Society of London, vol. viii.

registered 117° F. and in the other 110°, but the patient recovered.[1] Galbraith[2] has reported a case in which the thermometer registered 151°, and Jones[3] that of a girl, fourteen years of age, in whom the temperature rose to over 150°. In neither instance was the extraordinary heat attended with evil results. Duckworth reports[4] a case in which the thermometer marked 228° (108.9° C.). In all these extraordinary temperatures the possibility of deception practised by hysterical patients must be borne in mind. The temperature may be temporarily very high from emotion. I saw this once in a frightened child which had previously had but slight fever, and E. S. Tait has reported the same in the puerperal state.[5]

On the other hand, the thermometer may show a depression in temperature below the normal. The body heat often falls at the beginning of acute peritonitis. It is low after severe loss of blood, or if exposure to cold happen in alcoholic intoxication, during convalescence from acute diseases, and in melancholia. It is depressed by various poisons, and has been observed down to 93.9° in carbolic acid poisoning.[6] It is low in the insane. It may be only a fraction above 89° in the axilla in cholera. From any other cause it rarely, even in extreme collapse, sinks below 92°.

Though having its widest range of applicability in fevers, in other than febrile states, too, the thermometer assists greatly in diagnosis and prognosis. It is invaluable, in many instances, in discriminating between functional and organic affections. It aids in the study of apoplexy, of palsies, and of hysterical affections, and tells the true story in cases of feigned disease. It also enables us to judge whether increased frequency of pulse be due to fever or to debility; and it indicates that sweating which is not preceded by a previous elevation of temperature is the result of exhaustion and not of fever. There is a continuous rise of the heat of the body in all cases in which a deposition of tubercle is taking place actively in any of its organs, and more especially in the lungs; while, on the other hand, I have noticed that in cancerous affections the heat of the body is but little influenced, and is sometimes even below the normal standard.

Tongue.—When a patient is told to put out his tongue, it is not to see whether this organ is the seat of disease, but because experi-

[1] Philipson, London Lancet, April, 1880.

[2] Journ. Amer. Med. Assoc., March, 1892.

[3] Memphis Medical Monthly, Oct. 1891.

[4] Archives of Gynæcology, New York, Oct. 1891.

[5] Obst. Soc. Transact., 1884.

[6] Bäumler, in Quain's Dictionary of Medicine.

ence has taught that the tongue is a mirror, more or less perfect, of the condition of the digestive functions, and that it reflects the complexion of the nervous power and of the blood, and the state of the secretions. To judge of these varied circumstances, we have to examine the tongue in regard to its movements, its volume, its dryness or its humidity, its color, and its coating.

The *movements* of the tongue are impeded and tremulous in all conditions of the system attended with exhaustion. It is protruded slowly and with difficulty in fevers of a low type, and in nervous disorders which are accompanied by marked debility. The action of the muscles is seriously impaired in paralysis. In hemiplegia one side is crippled, and the tongue turns towards one of the corners of the mouth. When imperfect articulation is associated with difficulty in moving the organ, it commonly announces a serious cerebral lesion.

The *volume* of the tongue is changed by its own diseases; more rarely by the condition of the system at large, or by disturbances of the abdominal viscera. Yet a swollen or a broad and flabby tongue, on the sides of which the teeth leave their marks, is sometimes found in chronic ailments of the digestive organs, and as the result of the action of mercury, and of certain poisons. It is further observed in some affections of the brain, or as a consequence of a disturbed circulation attending diseases of the heart, and in distempers, like the plague, typhus, or scurvy, in which the blood is much altered. In affections of the stomach a flabby tongue showing marks of the teeth is a sign of decreased motility. The tongue is sometimes observed to be swollen on one side only in consequence of catarrhal inflammation. This hemiglossitis affects the left side, and is supposed to be of neurotic origin.[1] Loss of substance of the tongue, especially on its borders, is mostly due to syphilis. The ulceration is often associated with fissures.

Dryness of the tongue indicates deficient salivary secretion. In acute visceral inflammations, and still more frequently in febrile states, especially in the exanthemata and in typhoid fever, the tongue is dry; it may be so dry as to cause the papillæ to become prominent and the whole organ to appear roughened. The condition is one which, in acute diseases, is always to be dreaded, especially if the tongue be, in addition, of a dark color, glazy, or furred or fissured; for it is then a proof not only of generally arrested secretions, but also of depraved blood and of ebbing life force. Yet a fissured tongue is not, by itself, indicative of great and imminent danger; it may occur in

[1] Dyce Duckworth, Liverpool Med.-Chir. Journ., July, 1883.

chronic affections of the liver, or in chronic inflammation of the intestines; and in some persons it is congenital. In estimating dryness of the tongue we must not overlook the fact that this may happen from persistent openness of the mouth, as during sleep, from obstruction of the nasal passages, or from coma. Among chronic diseases the tongue is most apt to be found dry in diabetes. A dry, incrusted, brown tongue is due to a continuous crust on and between the papillæ, which is filled with parasitic growths. It occurs in states of prostration with lowering of nutrition and tendency to sinking. Dickinson has calculated that a dry tongue is present in about fifty per cent. of fatal cases; more than any other it foretells death.[1] The opposite of dryness, *humidity*, is, unless excessive, a favorable sign. It is extremely so if it succeed dryness, because it is a proof that the secretions are being re-established.

There is a rare disease of the tongue known as *xerostomia*, occurring in women after middle life, in which the dryness of the tongue is so extreme that it may prevent speaking or swallowing. The tongue is cracked like alligator skin, and looks like raw beef.[2]

The *color* of the tongue is subject to many variations. It is remarkably pale whenever the blood is watery and deficient in red globules. It is exceedingly red and shining in the exanthemata, especially in scarlet fever. The tongue is also very red if inflammation have attacked its substance, or the fauces, or the pharynx. It is bluish and livid when there is an obstruction to the flow of the venous blood or deficient aëration, as in some structural diseases of the heart and in dangerous cases of bronchitis or of pneumonia. A red, smooth tongue is a sign of failing nutrition. A tongue black in spots, the discoloration particularly marked about the middle of the dorsum, the papillæ enlarged, indicates a condition of parasitic origin.

As important as the color of the organ are the color and form of its *coating*. In health the tongue has hardly a discernible lining; disease quickly gives it one. In inflammation of the respiratory textures, at the beginning of fevers, in disorders of large portions of the abdominal mucous tract, the epithelium accumulates, and the tongue has a loaded, whitish appearance, due to excess of white epithelium on the papillæ with the intervals also more or less filled up. The coat is apt to be yellowish in disturbances of the liver, and of a brown or a very dark hue when the blood is contaminated. But we must

[1] The Tongue as an Indication in Disease, London, 1888.

[2] See reports of cases in Sajous's Annual of the Univ. Med. Sci., vol. i. 1891, C–1.

be sure, in drawing our inferences, that the abnormal aspect is not due to the food partaken of or to medicine. Its color is also modified by the character of the occupation. Thus, as Chambers tells us, there is a smooth, orange-tinted coating on the tongues of tea-tasters. A local cause sometimes gives rise to a thick, opaque coat. For instance, decayed teeth may produce a yellow sheathing on one side. Affections of the fauces also occasion a deep-yellow hue. Again, there are many healthy persons who wake up every morning with their tongues covered, more especially at the back, with a heavy coating, which wears off after a meal.

In some diseases the epithelium, which is either formed in excessive quantities or not thrown off, collects between the papillæ, leaving them uncovered and prominent. This is especially noticed in scrofulous children. When the epithelium is sticky and adherent, it winds itself chiefly around the filiform papillæ, elongating them and giving to the surface of the organ a *furred* appearance. Although this kind of tongue, as almost every other variety, is met with now and then in persons who are not ill, yet it may be generally looked upon as denoting disease. It occurs sometimes in chronic diseases of the abdominal viscera, but much oftener in grave acute maladies. The tongue, on the other hand, may be bare of its epithelium or imperfectly covered with it. We meet with this in certain instances of scurvy, or in cases of chronic diarrhœa and dysentery with great prostration, in which the tongue is often found to be red, smooth, and dry, or in attendance on cachexias, as the malarial. Again, a denuded tongue is common in scarlet fever, and not infrequent in typhoid fever. In scarlet fever it has a strawberry look. This is sometimes also seen in pneumonia.

The state of the *digestion* and the character of the discharges have so close a connection with the nutrition of the body that they become important general symptoms. But, for the sake of convenience, their value will be inquired into while discussing the diseases in the recognition of which they occupy the foremost place. A few words here, however, on the sensations of patients.

Sensations of Patients.—Sick persons are subject to many disagreeable feelings. They complain of chills, of heat, of languor, of restlessness, and of uneasiness; but their most constant complaint is of pain. Now, *pain* may be of various kinds; it may be dull or gnawing; it may be acute and lancinating. In its duration it may be permanent or remitting. A *dull* pain is generally persistent. It is most often present in congestions, in subacute and chronic inflammations, and where gradual changes of tissue are taking place

the pain of chronic rheumatism, and shades off into the innumerable aches of this malady. The only acute affections in which it is apt to exist are inflammations of the parenchymatous viscera and of mucous membranes.

Acute pain is in every respect the reverse of dull pain. It is usually remittent, and not so fixed to one spot. It is met with in spasmodic affections, in neuralgia, and, with extremely sharp and lancinating pangs, in malignant disease.

Pain varies much in intensity; it is sometimes so extreme as to cause death. We have to judge of its severity partly on the testimony of the sufferer, partly by the countenance, and partly by the attending functional disturbances. The latter are not to be overlooked, for they enable us, to some extent, to appreciate whether the torments are as great as they are represented to be.

The seat to which the pain is referred is far from being always the seat of the disease. A calculus in the bladder may produce dragging sensations extending down the thighs; inflammation of the hip-joint gives rise to pain in the knee; disorders of the liver occasion pain in the right shoulder. Pain felt at some part remote from that affected is either transmitted in the course of a nerve involved, or is sympathetic.

The same abnormal action does not always create the same kind of pain. Inflammation, for instance, causes different pain as it involves different structures: the pain from an inflamed pleura is not the same as that from an inflamed muscle. Speaking generally, the tissues themselves seem to determine the form of pain more certainly than does the precise character of the morbid process. Thus, pain in diseases of the periosteum and bones, no matter what may be the exact nature of the malady, is mostly boring and constant; in the serous membranes, sharp; in the mucous membranes, dull; and in the skin, burning or itching.

Pain produced by pressure is called *tenderness*. It indicates increased sensibility, and is most constantly associated with inflammation. Yet tenderness may be present without inflammation; the tenderness, for example, of the skin in hysteria. Commonly it is combined with pain occurring independently of pressure; but a part may be tender and not painful.

CHAPTER II.

DISEASES OF THE BRAIN AND SPINAL CORD, AND OF THEIR NERVES.

BEFORE entering upon a consideration of the affections of the nervous system it is proper to recall a few salient points connected with its structure and functions indispensable to a recognition of its derangements. We have constantly to bear in mind that there are in its composition nerve-cells composing ganglia, which are for the most part originators, and nerve-fibres, which are for the most part conductors, and besides, a peripheral termination of these conductors, which forms a peripheral nervous system, chiefly concerned in receiving and distributing impressions. Then, too, of late years much stress has been laid upon the nerve-cells, including the cell-body and its processes, and for each of these separate cell units the name neuron has been adopted. The most important process of the nerve-cell is the axis-cylinder, or the neuraxon. In the brain and spinal cord are the principal nervous centres which originate and control, and of the brain especially our knowledge of the subject of localization and special function of particular points has become so extended that it is made the basis of accurate diagnostic knowledge, which has of late years assumed the greatest practical importance.

Cerebral Localization.

A knowledge of the centres in the brain is a necessity for both diagnostic and surgical purposes. This knowledge has been acquired in part by experimental observations upon the lower animals, in part by clinical and pathological observations, and in part by electrical stimulation of areas of the cortex in the course of surgical operations upon human beings.

The localization of human cortical centres is indicated in the annexed sketch. It should not be forgotten that in all such diagrammatic representations the picture represents the fact but poorly. The two halves of the same brain are unlike. Moreover, there is never any hard and fast line dividing one centre from its neighbor. If they

do not actually overlap, the centres certainly pass into one another by indefinable gradations. The strength of the stimulus modifies the definiteness of limitation, and many facts go to show that the unaffected hemisphere has often a certain power of substitution, whereby it can take up the function of its injured fellow. It must be borne in mind that not muscles but movements have cortical representation, and that movements on each side of the body are represented in the cortex of both sides of the cerebrum, though in preponderant degree in that of the opposite side. On the other hand, there is, at least in the case of articulate speech, a location of the unique controlling centre singly upon one side or the other according as the person is right-handed or left-handed.

The prefrontal region—*i.e.*, that anterior to the motor area—is the seat of the higher mental processes. The movements of the lower extremities are represented in the upper fourth of both cerebral convolutions, the gray matter concerned extending in a mesial direction to the paracentral lobule, posteriorly to the superior parietal lobule and anteriorly to the first frontal.

Movements of the hip and knee are localized near the centres for the shoulder-movements; movements of the great toe somewhat above, at about the junction of the middle and posterior thirds of the leg area; movements of the other toes still farther back; movements of the ankle between the areas for knee and great toe. Movements of the spine and trunk are most strongly represented in the mesial aspect of the hemisphere in advance of the area for the movements of the lower extremity.

The cortical area governing the movements of the upper extremities occupies the middle two fourths of the central convolutions, extending posteriorly to the interparietal fissure and anteriorly to the frontal convolutions. This area contains from above downward subareas for the movements of the shoulder, elbow, wrist, thumb, and fingers respectively.

The area in which are represented the movements of the head occupies the lower fourth of the central convolutions, including the entire operculum, with the posterior portion of the third frontal and the dorsal lip of the fissure of Sylvius. The movements represented in this area, from above downward, are the orbiculo-palpebral, those of the angle of the mouth, and those of the lips and tongue. The movements of the platysma are probably represented in the posterior and inferior portion of this area. The centres for the movements of the larynx and pharynx are located in the anterior part of the lowermost portion of the anterior central convolution, and behind it is the

centre for the movements of the lower jaw. Those of the head and eyes are in the most anterior portion of the motor zone.

In the lowest portion of the ascending frontal convolution, and

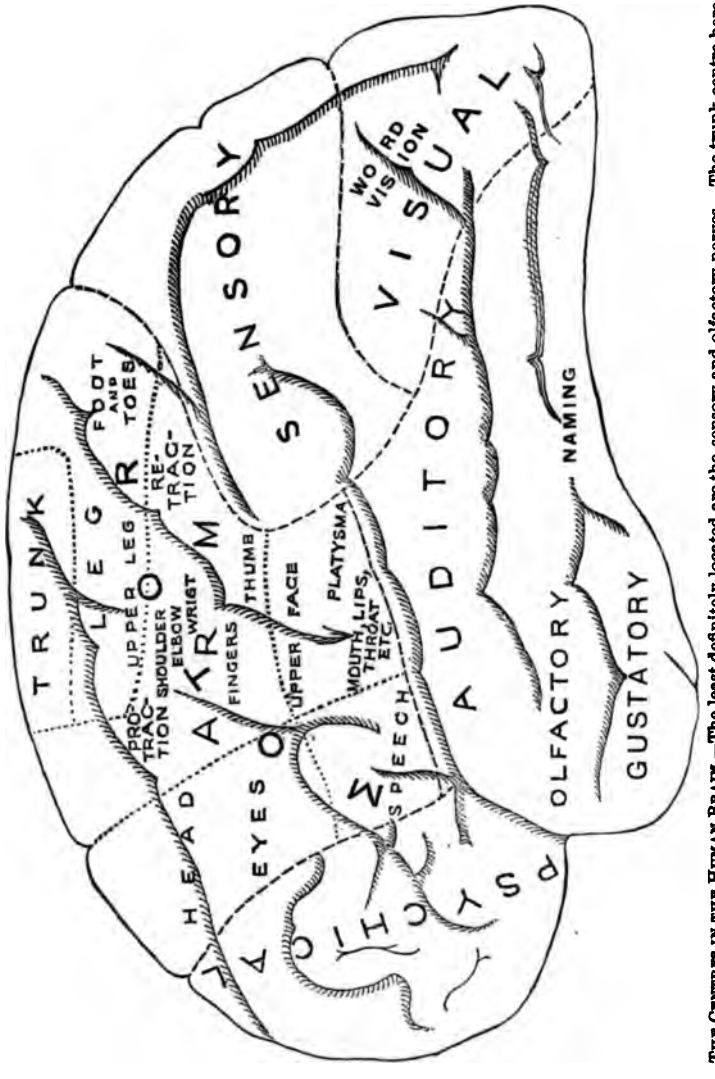


Fig. 9.

The Centres in the Human Brain.—The least definitely located are the sensory and olfactory nerves. The trunk-centre here indicated is also very uncertain. It is supposed to be located on the inner surface of the hemisphere, and the centre for the leg in the highest cordical part of the motor region, adjacent to the longitudinal fissure.

extending into the posterior portion of the third left frontal convolution, lies the centre for articulate speech, lesion of which causes motor aphasia. This is usually, though not always, associated with

inability to express thoughts in writing,—agraphia. But our complex power of thought-expression is made up of two other elements that are sensory; there must be psychical comprehension both of the heard and of the seen word. The centres intermediating these functions have been made out with some approach to definiteness. Lesions of the first temporal convolution produce word-deafness, or inability to comprehend the meaning of words though not deaf to other sounds. In the same way, word-blindness, or inability to understand the import of written or printed words, follows injury of the angular gyrus.

FIG. 10.

RIGHT HOMONYMOUS OR LATERAL HEMIANOPSIA, FROM LESION OF THE LEFT VISUAL CENTRE OF THE CORTEX OR LEFT OPTIC TRACT.—*A*, dark left nasal half-field from blind temporal half of retina; *A'*, dark right temporal half-field from blind nasal half of retina; *B*, left eye; *B'*, right eye; *C*, *C'*, left and right optic nerves, composed of the crossed bundles of fibres; *D*, *D'*, left and right crossed bundles; *E*, *E'*, left and right occipital lobes; *F*, *F'*, left and right posterior cornua; *G*, *G'*, "optic radiation" of Gratiolet; *H*, *H'*, optic chiasm; *I*, *I'*, angular gyrus; *K*, region of optic thalamus, geniculate body, and quadrigeminal bodies, collectively termed the primary optic centres; *M*, *M'*, cuneus of the occipital lobe, the cortical visual centre. The left cuneus and optic tract are shaded, to show lesion of these parts and the influence of the lesion upon the retinæ.

In reference to the cortical visual centre there can be little doubt that it is located in the occipital lobe, and especially in the cuneus. The production of hemianopsia from lesions of the occipital lobe, in accordance with the conclusions of Seguin,[1] is shown in the accompanying diagram (Fig. 10). Complete cortical blindness may be considered as a bilateral hemianopsia. The macula is also represented

[1] Journal of Nervous and Mental Diseases, 1886, No. 1, and Nov. 1887.

in the cortex. Dimness of sight in the opposite eye, with, as a rule, concentric diminution of the field, or crossed amblyopia, depends upon a lesion in the angular gyrus.

The auditory centre is most likely in the middle of the first temporo-sphenoidal convolution and related to the auditory nerve of the opposite side. The centre for smell is very probably on the medial surface of the temporal lobe at the anterior extremity of the uncinate convolution and in connection with the olfactory nerve of the same side. The cortical centre for taste is referred to the limbic lobe. The location of the centres for tactile or cutaneous sensation is also in dispute, but it appears probable that, if not identical with, they are at least contiguous to those of the motor functions of corresponding parts.[1] The muscular sense and the stereognostic sense seem to be represented especially in the cortex of the motor and parietal convolutions. A geographical centre, a centre for determining locality, is claimed to have its seat in the occipital lobe, near the visual centre; a naming centre has been located in the third temporal convolution, and a writing centre in the second frontal convolution. The psychical, or mental, processes have as their centres those parts of the cortex that have not been found to possess any special motor or sensory function, and particularly the prefrontal lobes.

It is often a matter of much importance, especially with reference to brain surgery, to determine on the skull the seat of the underlying cerebral centres. Broca, Horsley, and Reid have especially investigated the subject, and from their and other researches we are sure of these facts:

Under the frontal bone lie almost the entire frontal, middle, and about three-quarters of the upper frontal convolutions. The temporal bone covers the temporal lobe, except its anterior extremity and its posterior fifth. The occipital bone covers the greater part of the occipital lobe; the remainder of the cortex is beneath the parietal bone. The ascending frontal convolution starts somewhat lower that beneath the anterior inferior angle of the parietal bone in front of the prolonged line of the fissure of Rolando. In front of the precentral sulcus, the lower half of which is parallel to and behind the coronal suture, lies the root of the lower frontal; the root of the ascending parietal is behind the ascending frontal. The upper end of the fissure of Rolando corresponds to a point half an inch behind the middle of a line measured from the root of the nose upward to the occipital protuberance, and the fissure extends obliquely downward and for-

[1] See Dana, Journal of Nervous and Mental Diseases, Oct. 1888.

ward, at an angle of 67 degrees, to within a short distance of the fork of the Sylvian fissure. The fissure between the middle and lowest frontal convolutions is under the temporal ridge. The central convolutions are about an inch on each side of the fissure of Rolando; the centres for the leg, arm, and face lie on each side of the fissure. The angular gyrus is immediately behind the most prominent portion of the parietal eminence. The first temporal convolution is over the ear and mastoid process below the Sylvian line. This situation is determined by drawing a line from the external angular process of the frontal bone to a point three-quarters of an inch below the most prominent part of the parietal bone.

Sensory Centres, and Conducting Paths.

The sensory centres and the conducting paths by which the fibres unite the various parts of the brain, whether sensory or motor, and of the spinal cord, are not so definitely made out as the brain-centres have been; particularly uncertain are we as to the course of the sensory paths in the medulla, pons, and peduncle.

The sensory centres for the muscular sense and the sense of touch are supposed by Horsley and others to be in layers of cells in the motor cortex. But the centres for sensory impression are also claimed to be the hippocampal convolution and the gyrus fornicatus, and, generally, the occipital and temporo-sphenoidal lobes.

Volitional impulses originate in the motor cortex, and pass by converging fibres through the white substance of the hemisphere to the internal capsule, thence beneath the optic thalamus, to enter the crus cerebri, and through the pons, reaching the medulla, where the larger number of fibres cross to the opposite side of the cord to form the lateral or crossed pyramidal tract. The smaller fibres that continue onward form the anterior or direct pyramidal tract; these decussate in the cord at various levels. This constitutes the upper segment of the motor path of Gowers, which terminates in the ganglion-cells of the anterior horns of the cord. The lower segment consists in the fibres that originate in the efferent processes of the ganglion-cells and pass to their peripheral distribution in the muscles.

The fibres for the so-called cranial nerves leave the pyramidal columns as they approach the level of their nuclei on the opposite side of the medulla, to reach which they cross the median line somewhat in advance of the decussation of the remainder of the pyramidal tracts.

A lesion in any part of the upper segment of the motor path, between the cortical cells and the ganglion-cells of the anterior horns,

is followed by descending degeneration in the pyramidal tracts. The resulting paralysis is attended with increased reflexes, unchanged or but slightly changed electrical reactions, and little or no wasting of the muscles. A lesion in any part of the lower segment, between the gray matter in the cord and the terminations of the nerves in the muscles, gives rise to paralysis characterized by wasting, qualitative electrical changes, and impairment or abolition of the reflexes.

Sensory impressions reach the brain through the posterior roots of the cord, passing by the posterior and lateral columns in several tracts, most of which decussate in the cord. The sensory fibres for the muscular sense are supposed not to decussate in the cord, but in the medulla.

There is reason to believe that the paths for common tactile impressions, for painful impressions, for the conveyance of thermal impressions, and of the muscular sense, are distinct; that for the first coursing through the posterior column, those for the second and third through the antero-lateral ascending tract, and those for the last through the postero-median column and the direct cerebellar tracts.

Lesions of the peripheral sensory segment are attended, in addition to the impairment of sensibility, with abolition of the related reflexes. Lesions of the cord involving the posterior and lateral columns are attended with ascending degeneration in the postero-median and postero-external columns, the direct cerebellar and the antero-lateral ascending tracts.

Spinal Localization.

A centre for spasm is thought to be in the medulla at its junction with the pons, and is carried by the vagus; the cardio-inhibitory centre is in the medulla; the respiratory centre is in the medulla between the nuclei of the vagus and accessorius; the vasomotor centre is in the medulla; so is the sweat-centre in the medulla, with subordinate spinal centres.

The following facts will prove useful in localizing or determining the extent of a lesion of the spinal cord: Paralysis of the small rotators of the head and of the depressors of the hyoid bone points to involvement of the first and second cervical nerves; paralysis of the levator anguli scapulæ to involvement of the third cervical; paralysis of the sterno-mastoid, of the upper neck-muscles, and of the upper part of the trapezius to involvement of second, third, fourth, and fifth cervical; paralysis of the diaphragm to involvement of the fourth and fifth cervical; paralysis of the serratus, flexors of the elbow, and supinators of the forearm to involvement of the fifth and sixth cervical; paralysis of the shoulder-muscles to involvement of the fourth,

fifth, and sixth cervical; paralysis of the extensors of the wrist and fingers to involvement of the sixth and seventh cervical; paralysis of the extensors of the elbow, of the flexors of the wrist and fingers, and of the pronators of the forearm to involvement of the seventh and eighth cervical; paralysis of the lower neck-muscles and of the middle part of the trapezius to involvement of the sixth, seventh, and eighth cervical and first dorsal; paralysis of the muscles of the hand to involvement of the eighth cervical and first dorsal; paralysis of the intercostals to involvement of the dorsal nerves from the first to the tenth; paralysis of the lower part of the trapezius and of the dorsal muscles to involvement of the dorsal nerves from the second to the twelfth; paralysis of the abdominal muscles to involvement of the dorsal nerves from the seventh to the twelfth, and also the first lumbar; paralysis of the cremaster and flexors of the hip to involvement of the second and third lumbar; paralysis of the extensors of the knee, of the adductors, extensors, and abductors of the hip to involvement of the fourth and fifth lumbar; paralysis of the lumbar muscles to involvement of the second, third, fourth, and fifth lumbar nerves; paralysis of the peroneus longus, the flexors and extensors of the ankle to involvement of the fourth and fifth lumbar and first sacral nerves; paralysis of the flexors of the knee to involvement of the fifth lumbar and first sacral; paralysis of the intrinsic muscles of the foot to involvement of the first and second sacral; paralysis of the perineal and anal muscles to involvement of the third and fourth sacral nerves.

Loss of sensibility on the scalp points to involvement of the first, second, and third cervical nerves; on the neck and upper part of the chest to involvement of the second, third, fourth, and fifth; on the shoulder to involvement of the fourth and fifth; on the outer aspect of the arm to involvement of the fifth and sixth; on the radial aspect of the forearm and hand and on the thumb to involvement of the sixth and seventh; on the inner aspect of the arm, on the ulnar aspect of the forearm and hand, and on the tips of the fingers to involvement of the seventh and eighth cervical and first dorsal; on the front of the thorax to involvement of the dorsal nerves from the first to the tenth; over the ensiform cartilage to involvement of the sixth and seventh dorsal; on the abdomen to involvement of the dorsal nerves from the seventh to the twelfth, and also the first lumbar; at the umbilicus to involvement of the tenth dorsal; on the upper part of the buttock to involvement of the twelfth dorsal and first lumbar; in the groin and on the scrotum to involvement of the first and second lumbar; on the outer, anterior, and inner aspect of the thigh to in-

volvement of the second, third, fourth, and fifth lumbar nerves; on the inner aspect of the leg to involvement of the fifth lumbar; on the lower part of the buttock, on the posterior aspect of the thigh, and on the anterior, posterior, outer aspect of the leg and foot to involvement of the fifth lumbar and the first, second, and third sacral; on the perineum and about the anus to involvement of the third, fourth, and fifth sacral; and on the skin between the coccyx and anus to involvement of the fifth sacral and the coccygeal nerves.

Loss of the scapular reflex points to involvement of the fifth, sixth, seventh, and eighth cervical and first dorsal nerves; of the epigastric reflex to involvement of the fourth, fifth, sixth, and seventh dorsal; of the abdominal reflex to involvement of the dorsal nerves from the eighth to the twelfth, and also the first lumbar nerve; of the cremaster to involvement of the first, second, and third lumbar; of the knee-jerk to involvement of the second, third, and fourth lumbar; of the gluteal to involvement of the fourth and fifth lumbar and the first sacral; of ankle-clonus to involvement of the fifth lumbar and first sacral; and of the plantar to involvement of the first, second, and third sacral nerves.

Let us now look at the derangements of the nervous system. But first let us examine a few symptoms and morbid states having a general significance rather than a specific connection with any malady.

Temperature Variations.—These are not uncommonly induced by organic disease of the brain. Elevation may take place independently of febrile disease, as from irritation of the striate body or of portions of the cortex, and in conjunction with hemorrhage into the pons or medulla, vascular obstruction, and the epileptiform and apoplectiform attacks of general paralysis. The temperature is elevated also when infectious or inflammatory disease of the brain is present, such as tubercle or abscess, or meningitis. On the other hand, organic disease of the brain is often seen with subnormal temperature, for instance, extensive arteriosclerosis, old softening, and general paralysis.

Circulatory Phenomena.—Apart from febrile complications the pulse may be accelerated in disease of the medulla, or of degeneration in or about the vagus nucleus, or in consequence of irritation of portions of the cortex. Irritation of the vagus nucleus induces retardation of the pulse. This may result directly, as from inflammatory processes in or about the pons and medulla; or indirectly, as from increased intracranial pressure, such as attends hydrocephalus, hæmatoma of the dura mater, ventricular hemorrhage, brain tumor,

or from meningeal irritation or other reflex influence. The pulse frequency may be reduced to 40. Acceleration succeeding retardation is of unfavorable prognostic import.

Respiratory Disturbances.—These are observed together with coma, especially in disease of the medulla oblongata. The breathing may be accelerated and shallow, sometimes with intermissions; but more commonly it is slowed and deepened, and the pulse also is slowed. The breathing may be stertorous, or assume the Cheyne-Stokes type. In deciding that the respiratory derangement is from brain disturbance care must be taken to exclude disease of the lungs, of the heart, and of the kidneys.

Vomiting.—When of cerebral origin vomiting is unattended with pain, nausea, or retching, and while it may be induced by food, it often occurs independently of the taking and also of the character of the food. It may result through reflex influences, as from meningeal irritation, or through direct irritation of the vagus nucleus, from increased intracranial pressure and in coma. It is especially common in association with disease of the medulla, and particularly with tumors of the posterior fossa of the base of the skull.

The more direct symptoms of disorder of the nervous centres are manifestations of deranged intellection and deranged sensation.

DERANGED INTELLECTION.

The great instrument of the intelligence, the brain, manifests its disorders, whether primary or merely sympathetic, by derangement of thought of every conceivable degree and kind,—from dulness and confusion of the intellect to its utter perversion and prostration. When one intellectual function is disturbed, generally all are, or soon become so; yet we may find impairment of judgment and of imagination without deterioration of memory or of the powers of attention. One of the most marked signs of mental infirmity is an impaired memory. This is especially encountered in chronic cerebral diseases, or in such nervous affections of uncertain seat as epilepsy. Another signal of mental derangement is loss of judgment, or rather loss of power to appreciate the logical sequence of ideas; still another is depression of mind, or its opposite, exaltation. All these abnormal conditions may happen in acute as well as in chronic maladies, but they are more striking in the latter, and afford more aid in the diagnosis; and they may or may not be joined to appreciable textural changes. To the psychologist their significance is very great, as they are often the premonitory symptoms of that departure from mental health which terminates in confirmed insanity.

In acute disturbances of the brain delirium, stupor or coma, and insomnia are often prominent symptoms.

Delirium.—This is a wandering of the mind, manifesting itself by the expression of ill-associated thoughts, of the incongruity of which the patient is not conscious. It occurs most frequently in those of susceptible nervous system, and is more common in the young than in the old. It is almost invariably united with restlessness, and increases as night approaches.

The character of the delirium is various. There is first the *quiet* delirium, of a low or passive type. The patient mutters incoherent words, moans without any assignable reason, or lies silent, with his eyes open, his mind occupied with his vague illusions, and taking no notice of what goes on around him. If strongly aroused, he gives a rational answer, but not a long or a connected one, for he soon returns to his dreams and his ever-changing hallucinations. He picks at his bedclothes, moves in bed, and may even try to leave it, although he is easily prevented from so doing.

Then there is a delirium of somewhat more active type, still, on the whole, quiet; the patient wanders, yet not boisterously. He is irritable, and often does not show that his mind is disturbed, except in some one particular,—in irascibility about trifles, or in expressions and modes of thought foreign to his nature.

An active, *fierce* delirium presents different characteristics. The patient is wild, noisy; he sings, screams, gets out of bed; his face during the excitement becomes congested; the eye is bright, often fiery.

Now, all these forms of delirium occur in many different maladies, and are far from being of necessity linked to an organic cerebral affection. As a rule, we find the low, quiet delirium in conditions of vital exhaustion, particularly in those depressed states of the nervous system which are connected with quickened vascular action, and with a deterioration of the blood, as, for instance, in the low fevers. The fierce delirium may, however, be associated with prostration or depraved blood. Thus, the delirium of pneumonia is sometimes of a violent kind, owing to the maddening effect of the ill-oxygenated blood on the brain. In most of the ordinary fevers the delirium is of a moderate type; in inflammatory diseases of the brain and in acute mania it is fierce. The delirium of uræmia is apt to be active. If the delirium be due to cerebral disease, it is associated with headache; the headache of pyrexia generally disappears with the onset of delirium.

Delirium is not difficult of recognition; yet we must be careful

not to confound with it *night terrors*, those troubled dreams to which ailing children are so liable, and which occasion confusion of thought on first awaking, and until consciousness is fully aroused. Delirium is most likely to be mistaken for *insanity*. There is this palpable difference: an insane person is commonly in good health in all save his intellect; a delirious person is ill, and exhibits evidences of his illness besides his delirium. It is true that, when the patient is first seen, doubt may arise; but it is not of long duration. In the mania appearing occasionally after epileptic fits, or taking their place, there may be doubt until we obtain a clear history. Most perplexing are the cases in which insanity follows or attends inordinate drinking. But this is a subject which we shall discuss in reviewing mania a potu.

Another perplexing group of cases is furnished by the occurrence of that singular form of delirium which has been called the *delirium of inanition*, or of collapse. Its outbreak is sudden, like an attack of mania, but it is found to be combined with a feeble pulse, with a skin bathed in perspiration, with cold hands and feet,—in a word, with the signs of great prostration or of collapse. The seizure happens usually early in the morning, and is unexpected, for it occurs commonly at the end of the febrile state, and when the condition of the skin and pulse bespeaks convalescence. The exhausted nervous centre betrays itself in the sudden mental wandering, which has generally this characteristic,—there is but one fixed delusion, and this one connected with the subjects which have most engrossed the mind before the illness. The seizure lasts from six to forty-eight hours, and at its termination the patient is apt to awake out of a sleep with a calm mind, remembering, perhaps, his hallucination as a vivid dream. There may be more than one attack, but this is not common; and the duration is materially abridged by opium and by the employment of stimulants and nourishment. The form of delirium under consideration is not simply a sequel of defective brain nutrition in fevers. It may also succeed exhausting discharges and drains from the system, or inability to obtain or to digest the proper amount of food. Thus, it may happen in malignant diseases of the stomach; also in mere gastric irritability and persistent vomiting. The most marked instance of this kind of mental wandering I have encountered was associated with functional gastric disorder, which prevented enough food from being retained. In this patient the hallucination was on one subject,—a business matter which had been annoying him greatly just before his illness became decided.

Delirium is at times *simulated*. This differs from real delirium by

the absence of all other signs of illness, and by the sameness of the mental wandering. In a case of feigned delirium I met with, the man whined when spoken to, and pretended to rave; but his ideas always ran on the same subject, and he was very solicitous about his food, and about other matters of which a delirious person takes no notice. Delirium is more or less continuous; once delirious, a patient remains so for some time, and until the exciting cause subsides. In this respect hysterical delirium is exceptional; it does not last long, or it intermits and then reappears.

Derangement of Consciousness.—This may be of any grade, from simple clouding to complete loss. In the mildest degree, *somnolence*, the individual has an appreciation of his surroundings and can respond when addressed, sometimes intelligently.

In more profound impairment of consciousness, *sopor*, the individual lies half asleep and responds but sluggishly to sensory irritation, although he can be readily roused. He answers in monosyllables; is still capable of limited movement, and has a confused notion of his surroundings. Left to himself, he at once relapses into sleep, which is at times attended with mutterings.

A still more pronounced degree of impairment of consciousness constitutes the phenomenon called *stupor*. The patient lies in a deep slumber, from which he cannot be roused save with great difficulty, and when roused he answers reluctantly and briefly, and soon resumes his heavy sleep. The expression of his face is dull, yet now and then a ray of intelligence, excited by some object to which his attention is attracted or by some pleasant reverie, flits across his features. Swallowing is possible, and the reflexes are preserved, possibly exaggerated. Stupor is met with in several cerebral affections, and after an epileptic fit. It is also frequently seen in typhoid fever, or as the result of narcotic poisons. But there is nothing pathognomonic about it in these various conditions, nothing by which we can judge positively of its origin.

Coma is complete loss of consciousness: perception and volition are alike suspended, and there is an appearance of the profoundest sleep. The face wears a confused look; the pupils are sluggish and contracted or dilated; the mouth is open, the tongue dry. All conscious and unconscious response to sensory irritation is lost. Shouting or shaking will not arouse the individual. The extremities are relaxed and the reflexes are abolished. Swallowing is impossible, and the sphincter ani is no longer resistant. The breathing may be rhythmic, but it is frequently irregular; at times it is retarded and full, at other times of Cheyne-Stokes character: towards the close it

becomes stertorous and stridulous. Incontinence of urine and of fæces develops.

Coma always betokens a serious disturbance of the functions of the brain. It is often witnessed in cerebral lesions, as from pressure of blood or fluid in brain-substance or in ventricles, more rarely from tumors, abscesses, or thrombosis. The most complete coma is seen in apoplexy; it comes on quickly, and is attended with noisy respiration and a slow pulse. Another form of coma, scarcely less complete, is caused by narcotic poisoning; it, however, does not appear suddenly, and when from opium is associated with contraction of the pupils. Profound intoxication with alcohol induces coma, but the attendant symptoms, as a rule, make the association clear. The coma of fevers and of acute diseases, whether cerebral or not, is also gradually produced, but, unlike that due to the toxical effect of opium, is ordinarily preceded for days by insomnia, by delirium, and by other signs of cerebral disturbance. The coma of epilepsy is recognized by its following epileptic seizures. In the coma of Bright's disease the cause is made manifest by finding albumin and tube-casts in the urine, and by the evidences of preceding uræmia. Uræmic coma may, however, come on suddenly and pass off suddenly. It is, as a general rule, associated with dilated pupils. Coma also sometimes occurs in connection with diabetes. Under such circumstances examination of the urine will reveal the presence of sugar, perhaps also of acetone and of diacetic acid.

Sometimes a person appears to be comatose when his intellect is but little disordered. He may be paralyzed, and not have the power to communicate his ideas from crippled articulation or aphasia. This state is distinguished from coma by noting that the patient's attention is always directed to the questions asked him, nay, that he strives to answer them, but cannot; and that generally he has lost control over the muscular movements of one side of the body. Coma must not be confused with *syncope*, which depends upon cerebral anæmia, is usually of brief duration, and, except feeble heart action, is unattended with noteworthy symptoms.

Insomnia.—The deprivation of sleep is a concomitant of cerebral congestion and of the earlier stages of cerebral inflammation. But a person may be sleepless from excessive pain, from exhaustion, from grief, from mental excitement or fatigue, or from the too free use of coffee or of tea; sometimes insomnia is engendered by habitually working late at night.

Insomnia often precedes or attends delirium, as appears in typhoid fever. Among purely nervous affections it is most marked in delirium

tremens. It is a very troublesome symptom; but, occurring in so many abnormal conditions, it cannot be looked upon as having a distinct and specific diagnostic value.

DERANGED SENSATION.

The signs of perverted or impaired sensation are numerous. They may be either due to an alteration of the general sensibility or be the signals of a derangement of a nerve of special sense. Let us look at a few.

Hyperæsthesia.—An exalted sensibility of surface nerves—of those of the skin, the mucous membranes, or even of those of deeper-seated structures—may seem to be due to inflammation. We may, as a rule, distinguish the peripheral sensitiveness from the tenderness of subjacent inflammation by its extension over a larger surface; by deep pressure producing no more pain than a light touch; by the absence of signs of functional disturbance of the part involved apparently in inflammatory disease; by the uniformity of the painful sensation, no matter how long the duration of the disorder, though the sensitiveness exhibits distinct intermissions and exacerbations.

Hyperæsthesia is not closely connected with organic diseases of the brain or of the spinal cord. Indeed, it is in them not common, nor, as a rule, highly developed. By far the most usual causes of hyperæsthesia are impoverished blood and hysteria. Sometimes hyperæsthesia is produced by rheumatism or by gout, by lithæmia, or by disturbance of the function of the kidney. It is further met with in epidemic influenza; in hydrophobia; in inflammations in internal cavities involving the ganglia of the great sympathetic; after the use of ergot and of opium; and in some diseases of the skin. It also attends paroxysms of neuralgia, as witnessed in the exquisite sensitiveness of the skin during an attack of *tic douloureux*: the painful spots, too, in the course of local neuralgias are, when not the result of neuritis, hyperæsthetical. Hyperæsthesia attends the irritative stage of inflammation of sensory nerves from whatever cause.

The seat of the heightened sensibility is ordinarily in the skin, in the distribution of the cutaneous nerves. Yet hyperæsthesia may affect the nerves of the special senses, manifesting itself, for instance, by intolerance of light or of sound. But this variety of hyperæsthesia need here be but alluded to, as we shall presently look more fully at the signs of disturbance of these nerves. The increased sensitiveness may depend on irritation of the peripheral nerves, or of a cerebral centre, or of the conducting fibres of the spinal cord, especially of those of the posterior columns. Hyperæsthesia is often conjoined

to perverted sensation, and not a mere increase. When a painful sensation is more acutely felt than normal, it is called *hyperalgesia.* Sensibility to pain is most readily tested by a pinch or a prick, or by a wire brush with a faradic current.

Let us now look at hyperæsthesia in connection with affections of the nervous system, especially with those of the brain and cord.

Hyperœsthesia is general and combined with signs of organic disease.—We find this in tumors pressing upon the pons Varolii and corpora quadrigemina, or in alterations or injuries of the posterior columns of the cord and those producing irritation in the course of the conducting fibres, in some cases of cerebral meningitis, and in spinal meningitis in which the posterior nerve-roots are implicated. We have in all these conditions a hyperæsthesia more or less extensive, and often combined with hyperalgesia and with pain. In making up our minds as to the cause of the extended hyperæsthesia, the sensitiveness in diffuse neuritis, in general neuralgias, and in reflected irritation to the posterior columns, especially in hysterical subjects, must always be remembered.

Hyperœsthesia is limited to one side.—Limited hyperæsthesia belongs much more closely to spinal than to cerebral disease. We also find it in connection with special neuralgias, and the sensitive skin shows augmented electrical sensibility. In some instances of limited as well as of more extended hyperæsthesia nothing abnormal can be detected, and the disorder must be, with our present knowledge, set down as a neurosis, one concerning which it remains uncertain whether it be of central or of peripheral origin.

Anæsthesia.—Loss of sensation, or anæsthesia, is of various degrees. It may be complete or partial,—a perfect absence of sensibility or its mere benumbing. It may be of cerebral, of spinal, or of peripheral origin. It may involve only common tactile sensibility, or in varying combination and degree also the sense of pain, the muscular sense, the temperature sense, and stereognostic sense. In the parts affected with anæsthesia the nutrition is less active, the temperature is diminished, and there is a feeling of numbness. Frequently the circulation in the skin is retarded, occasioning a perceptible lividity and discoloration of the surface; or there are coexisting trophic changes, such as glazing of the skin and grayness of the hair. The electrical sensibility is diminished, and is made very manifest by the use of the wire brush with either the faradic or the galvanic current. In hysterical anæsthesia this is a particularly striking feature.

Loss of sensation has a much more constant connection with organic affections of the nervous centres than increased sensibility,

which, however, may precede it. In the insane, especially in monomaniacs, anæsthesia is common, and ordinarily very extended: so, too, in general paralysis. Indeed, with few exceptions, an *extended* anæsthesia points to an affection of the nervous centres. It may in these organic cases be both general and very complete.[1] *Localized* anæsthesia may be an early sign of degenerative change, and precede for a long time an attack of apoplexy with arteriosclerosis.

If the defective sensibility be owing to a spinal malady, it is generally found in the lower extremities, and coexists with paralysis. Anæsthesia of spinal origin is usually indicative of the sensory conducting paths in the posterior columns having been disturbed or altered; when about the body, as in transverse dorsal myelitis, there is mostly a zone of hyperæsthesia above the zone of anæsthesia. A limited area of anæsthesia, Allen Starr[2] has demonstrated, is caused by a limited lesion in the spinal cord, and the situation and shape of the area of anæsthesia tell us the level of the lesion. In hysterical paraplegia, in paraplegia from hypnotic suggestion, or that following railroad or other injuries, the line of lost sensibility is, as Charcot[3] has shown, very significant; it excludes the genital organs.

In accordance with the well-known law of the decussation of sensitive impressions in the cord, disease, if only of one posterior half, is followed by lost sensation on the opposite side of the body. One-sided anæsthesia, affecting even the face up to the middle line, is sometimes met with in hysterical subjects or after typhoid fever.[4] Hysterical hemianæsthesia is generally on the left side. But strictly limited one-sided anæsthesia is more apt to be found in a distinct brain lesion, and the particular affection occasioning the *hemianæsthesia* is disease of the white substance just outside of the optic thalamus, of the posterior part of the internal capsule, on the side of the brain opposite to the side of the body which shows the anæsthesia, or damage to the fibres which conduct sensation through the pons or the crus. A lesion involving the upper part of the pons may give rise to "crossed anæsthesia,"—namely, loss of sensibility upon the same side of the face and upon the opposite side of the body. Hemianæsthesia is a not uncommon symptom between the attacks of hystero-epilepsy.

A localized form of anæsthesia happens now and then in consequence of an affection of the fifth nerve. The extent of loss of

[1] As in a case reported by Winter, quoted in Schmidt's Jahrbuch, 1883, No. 1.

[2] Amer. Journ. Med. Sci., July, 1892.

[3] Œuvres complètes, iii.

[4] Calmet, Bulletin de la Société Médicale des Hôpitaux, 1876.

sensation depends much upon the part of the nerve at which the cause of disturbance is seated. The skin of the nose and cheek may become devoid of sensation; the reflex movements of the muscles of the face may cease; the conjunctiva, or the whole surface of the eye, or one-half of the tongue, may be deprived of sensibility. Only one of these phenomena, or all conjointly, may be encountered, according as part of one, or one, or all of the branches of the fifth nerve are affected. Sometimes, as Romberg proves, *trigeminal anæsthesia* is of rheumatic origin. When it is complicated with disturbed functions of adjoining cerebral nerves, it may be assumed that the cause is seated at the base of the brain.

Anæsthesia is stated to be sometimes the result of *reflex* action. It may thus arise in disorders of any of the viscera, and from an irritation of any sensitive nerve. It has, for instance, been observed in both lower limbs in sciatica. But in nearly all of these instances of supposed reflected nerve irritation there is really a neuritis.

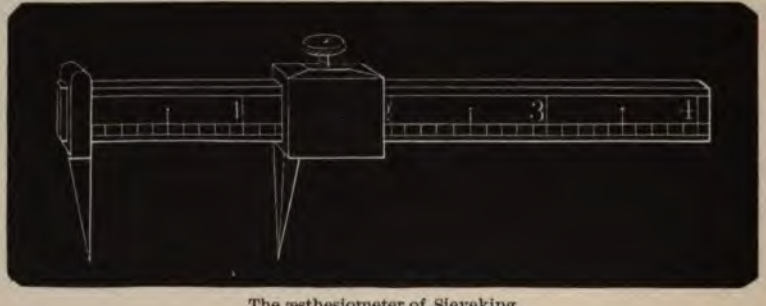
Diminished or lost sensibility to touch generally goes hand in hand with diminished or lost sensibility to pain, but the sensibility to pain may be augmented. This "anæsthesia dolorosa" is most commonly met with in multiple neuritis, and in spinal meningitis and myelitis from pressure.

Very often numbness and other altered sensations are complained of, and yet the whole is subjective; when tested, anæsthesia is not found. In endeavoring, indeed, to form an opinion of the existence or the completeness of anæsthesia, we do not trust to the patient's statements. We touch the part lightly with the finger or a feather while his eyes are shut, and the skin is pinched or a pin used to ascertain the extent of the impaired sensation. Or we resort to means by which we can make accurate comparisons; and one of the best is to pursue the method employed by Weber, which consists in determining how closely the points of a pair of compasses sheathed with cork may be approximated on the skin and yet be felt as two distinct points. An instrument for the same purpose, called the "æsthesiometer," was invented by Sieveking (Fig. 11), and is very much the same as the lighter one of Brown-Séquard now in common use. An instrument combining the principle of the beam compass with that of the mathematical one has been contrived by Ogle, and one with ivory points, by Manouvriez. In Carroll's æsthesiometer each arm is bifurcated, having one blunt and one sharp end, thus enabling us to test pain as well as touch. The points of the æsthesiometer, whether blunted or sharp, should be put down lightly and simultaneously, and parallel with the direction of the cutaneous nerves; at all events, the same

relative direction should be preserved in making comparative estimates.

To understand any results obtained regarding the tactile sense, it is necessary that we should be aware how this differs in some parts of the body. Most works on physiology contain an account of the researches of Weber and of those who have prosecuted the inquiry he started; yet a few of the conclusions may be here mentioned. At

FIG. 11.



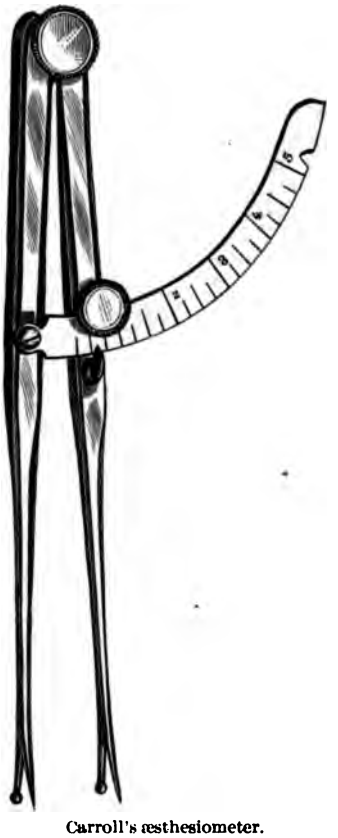
The æsthesiometer of Sieveking.

the tip of the tongue two points can be readily distinguished when separate from each other $\frac{1}{22}$ of an inch, or half a Paris line, one and a half millimetres; at the palmar surface of the third phalanx the limit is one line; on the palmar surface of the second phalanx, two lines, the same on the red surface of the lips; on the palm of the hand, the cheek, and the extremity of the great toe, five lines; on the back of the hand, at the knuckles, eight lines; at the lower part of the forehead, ten lines; on the skin over the patella and the dorsum of the foot, eighteen lines; over the middle of the arm, the thigh, and over the spine, thirty lines; on the back, sixty millimetres is common. But these observations are found to vary somewhat even in healthy persons, some being able to distinguish at a shorter distance than others.

Besides the impairment or loss of tactile discrimination, the altered sensibility may show itself in the loss of the faculty of feeling pinching, pricking, and other acts which excite pain, "analgesia;" or in insensibility to tickling; or in the want of appreciation of heat or cold, "altered temperature sensibility;" or in the loss of the sensation which attends muscular contraction, whether produced by the will or by an electrical current. Now, it is in individual cases always of importance to note which particular kind of sensibility is affected.

In sclerosis of the cord the sensation is retarded rather than lost.[1] A form of perverted sensibility, which may or may not be associated with anæsthesia, consists in the sensibility being more or less perfect, while there is doubt as to the side touched; indeed, the touch is commonly felt at a corresponding part of the other limb. This *allochiria*[2] is generally found in association with organic spinal disease; but it may also manifest itself in hysteria. A sufficient explanation of the erroneous reference of impressions is wanting. In a case recorded by Ferrier[3] the reversal showed itself also in the reflex reactions. Tickling the sole of one foot caused retraction of the other; tickling the inside of one thigh produced flexion of the other. Occasionally a single sensory impression is perceived as two or more; this is known as "polyæsthesia," and is most often met with in locomotor ataxia.

FIG. 12.



Carroll's æsthesiometer.

Sensibility to *temperature* has a close connection with sensibility to pain; but not always. There may be crossed paralysis of the thermal sense, while other senses are undisturbed.[4] Sometimes the temperature sense is exaggerated or diminished, or much perverted, and cold objects feel hot, and the reverse. Then points may be found in the skin where only cold, others where only heat, is appreciated. To test heat, a heated spoon or a test-tube filled with hot water is the readiest means; to test cold a sponge that has been dipped in cold water or a piece of ice is best.

Muscular anæsthesia has been mentioned. It is closely connected with the power we possess of estimating weight, the "muscular sense;" and the loss of ability to perceive differences in small weights, or the impairment of the sense of muscular movement and effort, is its most common form. It is really distinct from the sensi-

[1] Vulpian, Archives de Physiologie, t. i., No. 3.

[2] Obersteiner, Brain, July, 1881.

[3] Brain, October, 1882.

[4] Case reported by Weir Mitchell, Trans. Assoc. Amer. Phys., vol. vii., 1892.

tiveness of the muscles to pressure or to electrical stimulation, which may be also wholly wanting. The loss of the power of appreciating muscular contraction, as well as the deficiency of sensation, is most readily tested by the use of the faradic current; the contraction of the muscles produces no feeling.

Muscular anæsthesia is frequently combined with inability to determine the posture of a limb when the eyes are closed; it may or may not be associated with cutaneous anæsthesia. It is not uncommon in hysteria and in locomotor ataxia. Here the loss of the appreciation of the position of the limbs and of the sense of muscular effort is the usual variety. When the muscles are completely paralyzed, the muscular sense cannot be tested. The muscular sense has been localized by Allen Starr and McCosh at the junction of the superior and inferior parietal convolutions, behind the posterior central convolution.[1] In testing for the muscular sense, the eyes of the person on whom the test is made should be kept closed, and objects used should be of uniform size. To detect the difference in weight, and thus the resistance to contraction, Gowers[2] recommends leather balls containing weights from two drachms to two pounds. The weights are placed in a bag, suspended by a string to the parts to be tested.

The recognition of objects by the sense of touch, stereognosis, is not rarely impaired by lesions of the cortex, especially in the central and parietal regions.

Paræsthesia.—This is a perversion of sensation, not an exaltation. It does not disclose itself by pain and tenderness, but by itching, by formication, by unnatural feelings of various kinds, such as the feeling of tingling, of pins and needles, of goose-flesh, of thrilling, of flushing, of the trickling of cold water, of shock-like sensations, or of a sense of tightness, as in the girdle pain. It is generally purely subjective, though it may be influenced by touch. A form of paræsthesia is *acroparæsthesia*. This is chiefly characterized by numbness of the extremities. It is encountered in women at the menopause, and in those who do washing, scrubbing, or sewing. It may be also found in men, and by some is believed to be a neurosis, by others a neuritis.[3]

The alterations of sensibility discussed manifest themselves chiefly in connection with external impressions. Let us now look at some abnormal sensations which are not objective, but subjective,—arising

[1] Amer. Journ. Med. Sci., Nov. 1894.

[2] Diseases of the Nervous System, vol. i., 2d ed., 1893.

[3] Sinkler, Medical News, Aug. 1894, p. 178.

independently of external impressions. Headache and vertigo are of this character.

Headache.—In every case of headache we must first ascertain that the pain really originates within the cranium, and that it is not owing to supraorbital neuralgia; to rheumatism of the scalp; to disease of the bones; to periostitis, syphilitic or otherwise; or to affections of the ear. To accomplish this is generally not difficult. An inquiry into the history of the case, the locality of the pain, and its augmentation on pressure in most of the disorders named, furnish evidence which decides the source of the cephalalgia to be external to the cranium.

Another possible cause of headache, always to be kept in mind, has been made clear by the labors of eye-surgeons. It occurs in persons who have headache more or less intense, with abnormal sensations in the skin of the scalp, and at times vertigo and spasm of the eyelids and occipito-frontal muscle. The near use of their eyes increases their distress. When the eye is carefully examined, an optical defect is found, especially hyperopia or astigmatism. Again, we may have defective vision, with sleeplessness and severe headache, dependent on decayed teeth, and disappearing with their removal.[1]

Having settled that none of these conditions are present, we have to determine the probable cause of the headache,—a question the solution of which depends frequently more upon the symptoms attending the pain than upon its character. But let us glance at some of the common causes and characteristics of intracranial headache.

Headache is a rarely absent symptom of *disease of the brain.* In acute inflammation it is generally agonizing, and, while subject to exacerbations, continuous; it is associated with fever, with vomiting, and with delirium. In abscesses of the brain, in softening, and in similar affections which run a chronic course, the headache is less violent, and only occasionally paroxysmal; it is usually accompanied by signs of disturbed intellection and of deranged motion. In tumor of the brain the headache is apt to be severe and paroxysmal, but intellection is not at first much affected. In congestion of the brain the pain is dull, increased by stooping or lying down, by long sleep, and by bodily or mental fatigue; its concomitants are a flushed face, a throbbing of the arteries of the neck, an eye-ground in which the vessels, especially the veins, are turgid, and a heated head, with increased temperature, as shown by the surface thermometer. A form of congestive headache, apt to be relieved by bleeding at the nose, is

[1] Case reported by Ogle, Medical Times and Gazette, Aug. 1872.

often seen in young people at the age of puberty: the attacks are brought on by running or other violent exercise. In diseases of the meninges, especially those of a chronic character, the pain is constant and fixed, and sometimes very sharp. The latter kind of pain when persistent is also significant of disease of the superficial brain structures in contact with the meninges, and is usually felt at the place on the head which corresponds to the seat of the lesion within the skull.

Nervous or *neuralgic* headache is most common in women, especially in anæmic women. It is unremitting and very severe, yet of short duration; but after it is over there is great lassitude, and even some local soreness. It is not attended with rise of temperature, or with any signs of disturbance of the brain, except at times with a confusion of vision and an inability to carry on a connected train of thought. Anything that agitates the nervous system produces an attack; stimulants and food often relieve it. To the class of headache under consideration may be referred many cases of migraine.

But *migraine*, megrim, sick headache, or hemicrania, has certain symptoms which set it apart. The pain is usually attended with nausea and vomiting, is generally at first one-sided, and is accompanied, or more often preceded, by visual disorder, such as a bright spot gradually enlarging. The disturbance of vision begins suddenly, lasting perhaps for half an hour before the headache begins, and is at times associated with tingling on one side, with difficulty in speech and confusion of ideas; sometimes there is disturbance of hearing. The headache often begins in the temple, and is very severe; it spreads over the head, it may extend to the neck, or may leave the side originally affected to become agonizing on the other. There may be soreness of the head with the pain, and there is often pallor of the face, and a contraction of one pupil. Coldness of the extremities is not uncommon, and the patient vomits bile. This bilious vomiting often terminates the attack, which comes on only in paroxysms. Migraine is frequently met with in gouty or lithæmic persons, and the urine is of high specific gravity, and contains an excess of uric acid and urates, though, during the attack itself, no such increase may be met with. It is sometimes excited by reflex influences such as eye-strain, nasal or pharyngeal disease, dental caries, gastric disturbance, or uterine or menstrual derangement.

Sympathetic headache is found mainly in connection with disorders of the uterus and of the alimentary tube, and is often worse in the morning, before food has been taken.

Headache may be dependent upon various *poisons*, whether generated in the system or introduced from without; for instance, in

diseases of the kidney the retention of a large quantity of urea in the blood becomes the cause of persistent pain in the head. In torpidity of the liver, in lead poisoning, in opium eaters, in drunkards, after the use of strychnine or of large quantities of quinine, headache is common; and it is very likely that in persons with faulty assimilation certain ptomaines give rise to headache.

In studying headache as a symptom, we must always note what influence position and movements of the head, and percussion and palpation, have on the pain: whether, for instance, stooping, swinging the head from side to side, or rising rapidly from the horizontal to the erect posture affect it, and cause it to be combined with vertiginous or other abnormal sensations. In headache connected with organic disease of the brain the pain is increased by whatever increases the blood-pressure,—by stooping, by coughing, by any effort. The site of pain bears no definite relation to the site of lesion, except the lesion be near the surface. With severe paroxysms of pain vomiting often occurs. Headache increased by the erect posture and relieved by lying down bespeaks an anæmic condition of the brain.

Vertigo.—This is a transitory feeling of swimming of the head, a sense of falling, or illusory movements of external objects. The sensation is apt to occur whenever the circulation within the cranium is disturbed, and is often symptomatic of a disease of the heart, liver, kidneys, or of an affection of the stomach, or of gout or lithæmia; or it accompanies anæmia, or follows exhausting discharges. In the defective blood-supply to the brain, produced by arteriosclerosis, the vertigo, often very severe, is attended by signs of the morbid process in other parts of the body, and the tense pulse, increased blood-pressure, and accentuated second sound of the heart explain the cause of the giddiness. Extreme slowness of pulse, and a sensation of falling in a given direction, as in Ménière's disease, are not infrequent.[1]

Vertigo may attend any disorder of the brain. The *cerebral* form is recognized in part by the absence of those affections of other organs which would induce the dizziness,—and among these we must not forget eye-strain, and local palsies of the muscles of the eyeball, in part by its being joined to an almost constantly present sense of uncertainty in movement, to headache, and to further signs of an encephalic malady. Moreover, it is usually objective in character: surrounding objects appear to the patient to move, not he himself; and, unlike the subjective vertigo so common in mere sympathetic disturbance of the brain, closing the eyes relieves it.

[1] Grasset, Vertige Cardio vasculaire, Paris, 1890; Church, Medical N. 1892.

The most common form of vertigo, not arising from brain affection, is the so-called *stomachal vertigo*. It is apt to come on in paroxysms, sometimes in the middle of the night or in the early morning, and is associated with a dull, heavy ache in the head, and with more or less gastric disturbance, often following indiscretion in diet. Yet the tongue may be clean, and the digestive disorder so slight that it is only by the after-symptoms, by the relief afforded by attention to diet, and by remedies acting on the digestion, that we clearly make out the cause of the vertigo. Between the attacks the patient is free from the affection; though there are cases of more chronic kind, in which a certain amount of giddiness is present for long periods with only comparatively short intervals of freedom. The giddiness may become aggravated into a severe attack if the stomach be for a long time empty. In gastric vertigo there is no loss of consciousness. The pathology is obscure. Woakes[1] has endeavored to establish a direct nervous communication between the stomach and the labyrinth to explain the vertigo. Others regard the irregularity in the cerebral circulation produced by the gastric disorder, anæmia or hyperæmia, as the cause.

Very similar to gastric vertigo is the vertigo of malassimilation in connection with *lithæmia*. The history of the case, the state of the urine, the striking change which follows diet and treatment that alter the formation and elimination of uric acid, distinguish lithæmic vertigo.

Another form of vertigo of eccentric origin is that associated with partial deafness or ringing in the ears. Again, there may be an affection of the internal ear, the semicircular canals of the labyrinth especially being the seat of an inflammation, and the vertigo set in suddenly. Its onset is apt to be associated with vomiting, with suddenly developed tinnitus, with pain produced in the affected ear by the slightest noise, and with symptoms of apoplexy or a fainting condition. Such cases, to which Ménière particularly has called attention, may very speedily terminate fatally. But the acute seizure, which is by far the most common beginning of the *aural vertigo*, may leave behind giddiness and a persistent unsteadiness in standing and walking, or a tendency to go forward or backward, or a reeling gait. These, with the intense vertigo and the vomiting, the persistent noises in the ears, the unimpaired consciousness, and the deafness, become valuable signs of Ménière's disease. The deafness shows especially in defect of power of hearing vibration conducted through the skull. It is often one-sided, generally on the side of the marked tin-

[1] Deafness, Giddiness, etc., 1879.

nitus, and never absolute. Again, it may be noticed that there is deafness for certain groups of musical sounds, which Knapp accepts as proof that the disorder has extended to the cochlea.

In some instances the patient has a tendency to turn to one side or to walk round and round in a circle; and he is always miserable, although his general health suffers but little. The disturbance of the equilibrium is not always present; there may be disturbance of hearing without it. The vertigo is generally the most prominent symptom of the disease, and persistent vertigo not epileptic in character or obviously associated with an organic brain affection is nearly always aural. The dizziness is very apt to be severe, to come on in paroxysms, and to be excited by some effort or movement. It becomes associated with pallor, with faintness, with vomiting, and in part it remains even between the paroxysms. During these the roaring in the ears may or may not be increased, but signs of eye-disturbance are very apt to show themselves. The disease may result from any process that involves the labyrinth and the nerve-endings. It is more common in men than in women, and is very rare in young persons. It may come on after cold and exposure, or originate in gout or in syphilis. It has also been observed in men working under ground and breathing compressed air.[1] All cases of aural vertigo do not set in suddenly; some are slight, others are very severe and do not cease until the hearing is totally lost. Many cases progress slowly to recovery. Aural vertigo in its milder forms may be met with in affections of the ear that have had their origin in catarrhal inflammation travelling along the Eustachian tube.

A peculiar variety of *paroxysmal vertigo* has been observed in Switzerland, France, and Japan, associated with weakness of the extremities, drooping of the eyelids, and mental depression, but with preservation of consciousness. These attacks may occur very frequently or be months apart. They have been described by Gerlier, and the disease is known by his name, or as *paralyzing vertigo*. Toxæmia has been suggested as the probable cause.

To return to vertigo connected with cerebral or cerebro-spinal disease. There is a kind which Trousseau especially has described. The abnormal sensation is very short in its duration, but severe; the patient momentarily loses all consciousness. The vertigo recurs at uncertain times: while actively engaged, sometimes while in bed and half asleep. The head feels heavy after an attack, and the mind is temporarily stupefied; otherwise the health is good. This type of

[1] Curnow, Lancet, 1894, No. 3715, p. 1088.

vertigo is dangerous. It is often the *precursor of epilepsy*, and after a time becomes associated with convulsions.

Another kind of vertigo is that which arises from *overwork of the brain*, very likely connected with temporary hyperæmia. At times giddiness is the only symptom of disorder, and is present for many years, the patient enjoying otherwise excellent health. I have known a number of instances of this *essential vertigo* in which the tendency appeared to have been inherited. If it do not break out until late in life, it is a matter of more serious concern.

In *laryngeal vertigo*[1] there is a close connection with epileptic seizures. The chief symptoms are tickling or burning in the larynx, followed by vertigo, loss of consciousness, and spasmodic movements in the face and limbs. The larynx is healthy; but in a case observed by Sommerbrodt a polypus existed, removal of which cured the affection.

Allied to vertigo is the condition known as *astasia-abasia*, the most marked characteristic of which is difficulty in standing and walking. Consciousness is not lost, but sometimes there is a sense of giddiness. The affection is a manifestation of hysteria, of which other symptoms are likely to be present.

Besides headache and vertigo, there are various unnatural sensations, such as a feeling of momentary unconsciousness without giddiness; a feeling within the cranium of weight, of constriction; the feeling described as a rush of blood to the head; ocular spectra, and other false perceptions of many kinds and of every gradation. But I shall do no more than advert to this subject, and shall now consider some of the morbid phenomena of the special senses, particularly of the senses of sight and hearing.

DERANGEMENT OF SPECIAL SENSES.

Vision.—The sense of vision may be exalted, impaired, or perverted in disorders of the brain, whether organic or functional. It is exalted in inflammation; impaired, even totally lost, in softening, in tumors, in apoplexy, and during violent hysterical attacks simulating apoplexy. Perversions of the sense of vision are more frequent than its abolition, and probably more peculiar to cerebral affections. They are of all kinds,—some of great consequence, others of but little. *Muscæ volitantes*, or the appearance of spots and various small objects floating before the eye, have the latter significance; for they may happen in almost any form of cerebral disturbance, also in anæmia, in cardiac mal-

[1] Gasquet, Practitioner for August, 1878; Charcot, Progrès Médical, No. 17, 1879.

adies, in the neuroses, and in states of nervous exhaustion. They are simply the shadows of vitreous opacities or retinal vessels upon the retina, and have nothing to do with anything but the local condition, which is without significance. Of other manifestations of deranged sight, such as illusions, ocular spectra, and phantasms, I shall only state that they are more common in sick headache, and in derangement of the mind, temporary or permanent, than in recognizable organic disease of the brain. Yet they are found in affections of certain parts of the brain; for in disease of the posterior lobes, as Hughlings Jackson has observed, colored vision and optical illusions are frequent.

The *appearance* of the eye is often of as much significance as the derangement of sight. In some cerebral maladies the eye has a fixed stare; in others the eyelids are constantly moving: but the latter is a sign more frequent in chorea, local spasm, and hysteria. Great brilliancy of the eye is often noticed in meningitis and in insanity.

Derangements of the ocular mechanism may be the result of remote causes, or, themselves primary, may become the starting-point of disorder elsewhere. In the first case their study is valuable to the general diagnostician as indicative of the seat, nature, or stage of many diseases in other parts of the system; in the second case the diagnosis as well as the therapeutics of the distant and related disease is dependent upon the appreciation of the ocular derangement. It thus becomes evident that the abnormalities of the visual mechanism are of the highest importance in many systemic affections, particularly in disease of the cerebro-spinal system.

Let us first briefly consider the *idiopathic derangements of the eye that induce derangements elsewhere.* Both in origin and in result these are essentially functional. So far as relates to the eye they consist chiefly either in abnormalities of refraction, classed under the general head of ametropia, and comprising hyperopia, astigmatism, myopia, and presbyopia, singly or combined; or in incoördination of the external ocular muscles, commonly called insufficiency. The results of ametropia and muscular insufficiency are conveniently called *eye-strain;* and this condition generally evinces itself not so much in ocular or visual symptoms as in functional nervous derangements, often far removed and apparently disconnected. For example, it is a well-established fact that eye-strain is prone to produce headache, especially in young women after the age of puberty. These headaches are usually frontal, but may also be occipital, less frequently of the vertex or diffused. Eye-strain is at times the starting-point of choreic symptoms, and even, though this is rare, of genuine

chorea. Cases have been reported[1] showing that the same cause may produce functional gastric derangements, hysteria, melancholia, and even epilepsy. The lesson is obvious that when the origin of these or other functional affections is not otherwise explainable, we should at once proceed to exhaust the possibilities of a reflex neurosis due to ocular abnormality or to some other peripheral irritation.

Hyperopia and hyperopic astigmatism are much the most frequent sources of eye-strain, and by the aid of a mydriatic, followed by tests with the trial-lenses, the diagnosis of the existence and amount of the defect may be made. In the neurotic, or in those with intercurrent affections and weaknesses, the smallest degree may become the source of irritative strain. Muscular insufficiency is the next most frequent cause of ocular irritation. Simple myopia produces no strain, but myopic astigmatism, and presbyopia may sometimes cause it.

Turning now to the consideration of those *changes in the ocular mechanism* which indicate *effects and symptoms of disease elsewhere*, we find that disease in almost any part of the organism may give indications of its nature and location in the eyes. These symptoms, either singly or combined, are of a threefold nature:

Changes in the external appearances, and visible to the naked eye.

Changes in the fundus oculi, or eye-ground, as revealed by the ophthalmoscope.

Defects of vision as shown by the subjective report of the patient.

The first and last set of symptoms require no very considerable special training to study, but the use of the opthalmoscope does demand it, and often to such a degree that many are compelled to forego invaluable knowledge, unless they can avail themselves of the services of an expert.

1. Among the *external abnormalities of the eyes* exception must, of course, first be made of such local diseases as have no systemic relations, such as ecchymoses, congestions or inflammations of the lids and conjunctiva, trachoma, glaucoma, cataract, congenital anomalies, etc. Herpes zoster ophthalmicus, a peripheral neuritis of the ophthalmic branch of the fifth nerve, is a dangerous and painful malady, often if not always, owing to local causes. Exophthalmos is either due to local disease or is present as one of the main symptoms of the affection called exophthalmic goitre. Diseases of the nucleus or of the ganglion of the fifth nerve or of its ophthalmic division may result in inflammation [illegible] of the eyeball.

[1] [illegible] Nerves. By Weir Mitchell, [illegible]

Next in importance is a class of diseases due to external infection that generally points to a source of contagion elsewhere in the organism. Cases of localized tuberculosis of the conjunctiva have been reported wherein the handkerchief has perhaps carried the bacillus to the eye. Gonorrhœal ophthalmia is a constantly recurring disease in ophthalmic practice; but the most frequent and frightful is the ophthalmia of the new-born,—ophthalmia neonatorum,—due to infection during labor with the vaginal discharges of the mother. It is said that the greater part of the blindness of the world is due to this wholly preventable disease.

Affections of the conjunctiva or lids may have their origin in diseases of the adjacent skin or mucous membrane, and extend to the eyes by simple contiguity of structure. A close connection frequently exists between hay-fever, catarrhal and other diseases of the nasal mucous membrane, and similar conditions of the conjunctiva.

Arcus senilis, a ring of grayish tissue-change about the corneal limbus, betokens generalized atheromatous or fatty degeneration, chiefly arterial or cardiac. Interstitial or diffused keratitis is nearly always the result of inherited syphilis.

Of the remaining affections of the external parts of the eye indicative of general or internal disease, the most important are those pertaining to the muscles of the eye or movements of the globe. They easily fall into two groups,—those of the external and those of the internal muscles.

Strabismus, or squint, may be due to local causes, such as injuries, or cold, etc., but it usually arises from a lack of equal or balanced power among the twelve external muscles, and to ametropia and anisometropia. The distinctive subjective characteristic of squint is double vision. In examining for strabismus we observe whether the eyeball is turned inward or outward. In paralysis of the external rectus we have ordinarily an internal or convergent squint, in paralysis of the internal rectus an external or divergent strabismus. In palsy of the superior rectus there is inability to raise the eyeball in a proper manner above the horizontal level; inability to lower it below indicates palsy of the inferior rectus. Strabismus due to local causes must be distinguished from true paralytic squint due to more centrally located lesions. It must also be distinguished from spastic action of the muscles caused by irritative intracranial injuries. In both the latter cases there is a conjugate or common movement of both eyes to one side or to the other, called *conjugate lateral deviation;* the head often shares in the lateral movement. In spastic irritative lesions of the cortex the eyes are turned from the side of the injury;

in paralytic or destructive lesions they are turned towards it. The eyes, as has been said, look at the lesion in paralysis, away from it in spasm. The symptom, however, owing to its frequently temporary existence, and also to the fact that it may arise as an indirect symptom, must not be relied upon except in conjunction with others and when continuing for several weeks.[1] The seat of the lesion may be in the cortex, the internal capsule, or the pons; in the latter case the symptoms are direct and the deviation of the eyes is the reverse of that given above: the eyes in paralysis look away from the lesion; in spasm, towards it. If in lesions of the pons the sixth nerve nucleus be included, there is, of course, paralysis of the external rectus, so that the corresponding eye cannot be rotated outward past the middle line, whilst the other eye cannot be rotated inward past the middle line. This associated movement of the other eye will not be impaired if the injury to the sixth nerve be between the nucleus and the globe.

Owing to the peculiar position of its nucleus and the long course of exit of the *sixth nerve*, its exclusive paralysis is the most frequent of single nerve palsies. It is peculiarly liable to paralysis from indirect or pressure causes, but if connected with paralysis of the opposite side of the body and with other symptoms of brain disease, it clearly points to a lesion of the pons. In consequence of the close anatomical relations of their nuclei, palsies of the sixth and facial nerves are frequently associated. Other nerves originating in the pons are liable to implication. Next to the sixth the *third nerve* is the one most often paralyzed, and in proportion to the number of twigs involved and the completeness of their palsy is there a probability of a lesion at the base of the brain. The various paralyses of the external ocular muscles are usually attended with double vision, *diplopia*.

Ptosis may exist either with or without involvement of other third-nerve branches, but in any case the value of the droop of the upper eyelid as a localizing symptom is somewhat indeterminate. If of one eye alone, ptosis usually indicates a cortical lesion, unless due to evidently local causes. In paralysis of the third nerve we have, besides the ptosis, dilatation of the pupil of moderate extent. Inability to close the eyelids is associated with paralysis of the facial nerve.

As regards the nature of the lesion, the ocular symptoms generally give little definite indication.

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[1] The direct symptoms are those intimately dependent upon the lesion of a part; the indirect or distant symptoms are those due to disturbances of circulation, to pressure, to the reflex or inhibitory effects at other points than the seat of injury.

Abnormalities of the pupils are understood by remembering that the third nerve controls the contractile mechanism and the cervical sympathetic the dilating mechanism. Hence an unusual diminution or increase of either innervation, especially of the first, causes alterations of the pupils at once. Irritative cerebral lesions thus produce contraction, whilst lesions which destroy cerebral function produce morbid dilatation. The state of the pupil in tumors, hemorrhage, and inflammatory conditions of the brain may thus furnish us with most serviceable indications of the extent and destructiveness of the injury. When but one pupil is abnormal, the rule above given serves to indicate lesion of the corresponding half of the cerebrum, irritative or paralytic according to the degree of the injury. Yet one-sided contraction, like one-sided dilatation, may also be owing to tumors at the root of the neck. Hemorrhage or effusion into the pons or lateral ventricles, when small or irritative, produces contraction; but if large, permanent dilatation. Certain drugs, such as opium, contract the pupil; belladonna, chloral, and cocaine dilate it. We also find dilatation of both pupils in chlorosis. If the foot be pricked, the pupils dilate, provided the iris be uninjured and the sensory columns be intact. In epileptics this reflex excitability is greatly diminished.[1]

The pupillary reaction to light may be useful in diagnosticating the location of a lesion, whether beyond the corpora quadrigemina or not. If beyond, the pupillary reflex will be retained, despite the loss of sight. Lesions of the spinal cord and of the sympathetic nerve produce results the reverse of cerebral disease: irritative lesions dilate, paralytic lesions contract. In this connection the Argyll-Robertson pupil—the light-reflex lost, the accommodative reflex retained, of a myopic pupil—is of value as indicating, often early, sclerosis of the posterior columns of the cord. When hemianopsia is due to disease of the optic tract, the pupil fails to react to the stimulation of light reflected upon the blind half of the retina; contracting, however, if the lesion be situated in the cerebral hemisphere. *Paralysis of the accommodation* may exist independently of pupillary involvement, and its significance is that of paralysis of other branches of the third nerve. Paralysis of many or of all the muscles of both eyes, *ophthalmoplegia*, is usually due to a lesion of the nuclei of the supplying nerves. It is especially due to lesions of the nuclei in the gray matter of the Sylvian aqueduct.

II. *Abnormal changes in the fundus of the eye* may be of great

[1] Lawson, West Riding Reports, vol. iv.

diagnostic value, and in almost every case of circulatory or nervous disease the ophthalmoscope gives valuable hints concerning the general disorder. With few exceptions these changes are symptomatic, and do not arise from local disease.

We should invariably examine with the ophthalmoscope the eyes of patients suspected of having disease of any part of the cerebro-spinal nervous system. Changes in the eye, indeed, often occur early enough to be the first certain sign of disease, and this, too, without any impairment of sight; on the other hand, lesions indicating cerebral or other organic affection have been found in cases in which failure of sight was alone complained of. But particularly is the ophthalmoscope valuable in enabling us to differentiate organic from functional affections. It tells us of extension of congestion or of inflammation of the brain to the internal structures of the eye, or of the amount of resistance offered to the circulation within the cranium. This resistance may either arise from a marked "coarse" lesion, or may make itself felt through the sympathetic nervous system.

The changes in connection with organic disease have been observed chiefly in the retina, the optic disk, and the choroid. In using the *ophthalmoscope for medical diagnosis* we pay particular attention to these structures; especially do we note the disk, its color and size, and the pigment around its edges, the region of the macula, the size and appearance of the arteries and veins, whether diminished, enlarged, or tortuous, whether there are exudations or hemorrhages in the course of the vessels, and in what part of the eye-ground the patches are most marked.

Hyperæmia, or increased redness, is due to local causes; and the fundus changes in myopia, astigmatism, retinitis pigmentosa, and some forms of choroiditis are also to be excepted. In *diseases of the blood* and the blood-making organs, the indications are remarkably clear. *Retinal hemorrhages* are a common concomitant of such general diseases as albuminuria, diabetes, anæmias, cardiac valvular disease, arterial, atheromatous, and fatty degenerations, chronic malaria, and other febrile conditions. *Embolism of the central artery of the retina*, causing unilateral blindness, points to cardiac valvular disease. There is a grayish discoloration about the macula, with a central cherry-red spot. Tortuosity, beading, bulging, and irregularities in the size of the arteries, with pressure on the veins, œdema, and hemorrhage, are indicative of arteriosclerosis. *Simple anæmia* is at once recognized by the transparency of the blood-columns, and *lukæmia* and *pernicious anæmia* produce characteristic changes in the eye-ground, especially the last, with retinal œdema and hemorrhages,

disk-discoloration, arterial pallor, and venous distention. *Albuminuric retinitis* is common, but not invariable in albuminuria. The typical fundus changes consist in an early stage of haziness of the papilla and central part of the fundus, slight hemorrhages, and faint grayish discolorations. Later, white dots or splotches are grouped about the macula, or, flame-like, radiate from it. Striate hemorrhages are scattered over the fundus, the papilla is œdematous, and its limits are obscured. The ophthalmoscopic signs of *diabetic retinitis* are very similar. Visual disturbances, however, do not, in either case, stand in any exact ratio to the defects of the eye-ground.

Atrophy of the optic nerve, recognizable by the whiteness or discoloration of the disk, failure of vision, even to blindness, may sometimes seem to have no remote causes, but is commonly associated with, or is a result of, diseases or lesions of the spinal cord or the brain, toxic substances in the blood, papillitis, etc.

Papillitis, optic neuritis, "choked disk," is a symptom of most decided diagnostic value. The picture is easily recognized, consisting in a swollen red disk, the edges and vessels of which are obscured by a "woolly," striate blurring extending to the adjacent retina. This condition is always symptomatic, and in the large majority of cases points to tumor of the brain, though other intracranial diseases may produce it. From papillitis, however, nothing can be argued as to the nature or location of the tumor or other affection. It is often not a late symptom, and unimpaired vision may coexist. Optic neuritis has been observed after measles and scarlet fever, also after malaria and typhoid fever.[1]

Choroidal inflammations are chiefly distinguishable by the striking color and pigment changes of the fundus. Plastic choroiditis is commonly secondary to meningeal affections and prostrating fevers; purulent choroiditis, to local or general infection or septicæmia. Disseminated and central choroiditis, or choroido-retinitis, is frequently the result of syphilis. The choroid is peculiarly liable to become the seat of tuberculous growths.

III. Passing now to the consideration of purely *subjective visual derangements*, it becomes highly necessary to determine first whether such defects are due to refraction-errors, insufficiencies, and other local causes, or if they are secondary and symptomatic. Unless other indications are present, the complaint of headache, especially if frontal, weariness or pain of the eyes after near work, affections of the lids and conjunctiva, conjoined with general irritability and func-

[1] White, Journal of the Amer. Med. Assoc., Oct. 1893.

tional gastric derangements, almost invariably indicate eye-strain as primary. Simple inability to see distant objects clearly, without other symptoms, local or general, indicates myopia.

Amblyopia, due to the excessive indulgence in tobacco or alcohol, has but a single objective sign: an unusual pallor of the temporal portion of the papilla. There is deterioration of visual acuity, to which subnormal color-perception may be added. Amblyopia sometimes occurs also as a manifestation of hysteria and in association with migraine. It has further been observed as a symptom of intoxication with quinine, iodoform, lead,[1] or after sexual excesses; or the defective acuteness of vision shows itself as a day-blindness or as a night-blindness; or takes the form of contracted fields of vision, or of color-blindness. Marked visual deterioration of a single eye should lead to inquiry for extra-local causes. It may be due to disease of the corresponding optic nerve. When ametropia has been excluded and the above-described ophthalmoscopic signs are wanting, the cause must be sought in disease of other organs. Paresis, and even paralysis of the accommodation, and visual failure, are not infrequent as *reflex neuroses* from peripheral irritation of other parts. Cases of abnormalities of dentition and other dental troubles producing such visual defects have been frequently reported. Menstrual difficulties, masturbation, the influence of pregnancy and lactation, may sometimes account for obscure ocular troubles. Hemeralopia, night-blindness, due to deficient nutrition of the general system, has been traced to insufficient food.[2]

Modifications of the color-fields have been found chiefly in hysterical patients. The field for red and green, always the narrower, shows the restriction most markedly.

The most important ocular sign of cerebral disease, and one invariably pointing to intracranial affection is *hemianopsia*, or loss of vision of the halves of the fields. The most common variety is that called homonymous lateral hemianopsia, in which the loss is either of the temporal half of one eye and of the nasal half of the other, or *vice versa*, a vertical line nearly through the centre being the dividing line. There are three other forms of hemianopsia, called temporal, nasal, and altitudinal, in which the half-fields are respectively the two temporal, the two nasal, with the dividing line, as previously, perpendicular, or the two dark half-fields are the upper

[1] De Schweinitz, The Toxic Amblyopias, 1896.

[2] Kubli, Archiv für Augenheilkunde, June, 1887, who describes three hundred and twenty cases occurring during the Russian church-fasts.

or the lower halves, with the dividing line horizontal. These three varieties are seldom met with, and, from the peculiar anatomical relations of the optic chiasm or commissure, are readily recognized as the results of lesions of this part, either at one side or the other, above or below. Homonymous lateral hemianopsia always indicates lesion beyond the chiasm. If the hemianopsia be "relative,"—involving only a part of the perceptions of light, form, and color,—it must necessarily proceed from a partial lesion of the common visual centre situated in the cuneus of the occipital lobe.[1] But if the hemianopsia be absolute,—with complete loss of light, form, and color sense,—the lesion may be either one affecting the entire visual centre of one side, or one rendering wholly functionless the fibres of one radiation, internal capsule, or optic tract. If the latter were the case there would almost certainly be other intercurrent or general symptoms, such as paralysis of other cranial nerves, hemianæsthesia, some form of aphasia, or hemiplegic symptoms. A symptom of great value in locating the lesion of hemianopsia is the hemiopic pupil. Convergence of a narrow cone of light upon the insensitive half of the retina yields no pupillary reflex if the lesion be in the optic tract; if the pupil, under such stimulus, contract, the lesion must be beyond the tract. The intracranial affection giving rise to the hemianopsia may be of malarial origin, and it and the associate cerebral symptoms will disappear under active antimalarial treatment.[2]

Mind-blindness, physical vision, but failure to realize the psychical import of the things seen, sometimes a symptom of general paralysis and obscure cerebral disease, indicates a cortical lesion in the occipital or occipito-temporal lobe, near by if not conterminous with the visual centre.

Hearing.—As regards the *sense of hearing*, the same may be said as of vision. It, too, is perverted and impaired in various cerebral affections. Yet, to be certain that the cause of the difficulty is cerebral, the ear must first be examined with reference to any physical imperfection; and in doing so we may by means of the otoscope get an idea of the vascularity of the drum, and be led from this to infer the condition of the vessels of the brain. We must also examine the throat and the condition of the Eustachian tube, for catarrhal inflammation extending to the middle ear may give rise to a form of aural vertigo.

[1] Seguin limits the centre to the cuneus; Nothnagel makes it include also the posterior portion of the superior occipital convolution.

[2] See my paper on Malarial Paralysis, with eye examinations by Harlan, in International Clinics, vol. iii., Ser. I., Oct. 1891.

Great acuteness of hearing and intolerance of sound are generally symptoms of extreme nervous irritability, or of beginning cerebral inflammation. Deafness may be owing to softening of portions of the brain; but Ferrier tells us that it is not met with in destructive lesions of the cortex. Deafness is also found as a temporary and by no means unfavorable symptom in the continued fevers. Imaginary sounds and ringing noises in the ear, or *tinnitus aurium*, are frequent accompaniments of cerebral disorders. But the latter is encountered in so many different conditions—in diseases of the cerebral vessels, in congestion of the brain, in Ménière's disease, in affections of the heart, in anæmia—that it is a sign of little moment; and, in truth, its most usual cause is local,—namely, an accumulation of wax in the meatus.

There is a form of reflex, the so-called *binaural reflex* described by Gellé, the disappearance of which is of value, provided we have been able to exclude disease of the ear. In health, when a vibrating tuning-fork is placed before one ear, while pressure is made by means of a Politzer bag on the canal of the other, a diminution of the sound of the fork is noticed. In disease of the cervical cord this reflex disappears.

DERANGED REFLEXES.

Derangement of the reflex action plays a most important part in the study of diseases of the nervous system. Each action is brought about by a sensory nerve that conveys the impression to the centre, by a motor nerve that transmits the impulse from the centre to the periphery, and by a reflex centre between the two in the spinal cord connecting the roots of the sensory and motor nerves, which with them forms the "reflex arc." The reflex centre is to some extent under brain control.

There are two forms of reflexes to be especially studied,—the cutaneous or superficial, produced by stimulating the skin, and the deep reflexes, the muscle or tendon reflexes, evoked by tapping muscles or tendons.

The *superficial* may be almost everywhere excited by tickling or gently stimulating the skin. The most usual ones to be noted are the reflex of the sole of the foot, the *plantar reflex;* and that of the palm of the hand, the *palmar reflex*. The former, when normal, attests the integrity of the reflex arc at the lower end of the cord; the palmar reflex, contraction of the digital flexors by tickling the palm, indicates a normal state of the reflex arc through a greater part of the cervical enlargement. Other superficial reflexes which may be mentioned are the *cremaster reflex*, the drawing up of the testicle excited by stimu-

lating the front and inner side of the thigh, and originating in the cord at a point between the first and second lumbar pairs; the *gluteal reflex*, the contraction caused by irritating the skin over the buttock, and showing the integrity of the cord at the fourth and fifth lumbar nerves; the *abdominal reflex*, a contraction in the abdominal walls caused by scratching the skin on the side of the abdomen, and depending on the action of the cord from the eighth to the twelfth dorsal nerve; the *epigastric reflex*, an epigastric dimpling produced by stimulating the side of the chest in the fifth or sixth intercostal space, and indicating integrity of the cord from the fourth to the seventh pair of dorsal nerves; the *scapular reflex*, a contraction by stimulation of the scapular muscles, and bespeaking the integrity of the reflex arc at the level of the upper two or three dorsal and lower two or three cervical nerves; the *erector spinæ*, showing itself by stimulating the skin along the border of the erector spinæ muscle, the contraction of these muscles showing the healthy state of the cord in the dorsal region. Other reflexes of indeterminate utility are the *platysma reflex*, dilatation of the pupil upon pinching the platysma myoides muscle. Among *cranial reflexes*, the more noteworthy are the *iris-contraction* upon exposure of the retina to light; the *eyelid-closure* from irritation of the conjunctiva; the *pharyngeal*, *laryngeal*, and *palatal reflexes* (cough, swallowing, etc.) from irritation of these parts; and *nasal reflexes*, as in sneezing. The *aural reflexes* are of some value in appreciating disease of the cervical part of the cord.[1] In disease these superficial reflexes are often absent. Thus, disease of one cerebral hemisphere diminishes or destroys them on the other side, the paralyzed side of the body. In pregnancy all reflexes are increased. The superficial reflexes are much influenced, increased or diminished, by psychical causes.[2]

The reflex phenomena connected with the tendons give us the best illustration of the so-called *deep reflexes*. The tendon of the patella is the one most readily studied; and if, the body being bent forward, we strike abruptly the tendon of the patella just below the knee-cap, after rendering the ligamentum patellæ tense by flexing the knee at a right angle while one knee-joint rests upon the other, or the leg hangs loosely over a supporting arm, a sudden contraction takes place in the quadriceps femoris muscle, and the foot is jerked upward. When very slight, the knee-jerk is most readily elicited by a tap with the percussion hammer. This reflex is largely due to a

[1] Amer. Journ. Med. Sci., Dec. 1888.

[2] Jendrassik, Deutsches Archiv für klinische Medicin, April, 1894.

muscle reflex action dependent upon the spinal cord. There are several instruments for measuring the knee-jerk. A good one is that of Hayne's.[1]

The knee-jerk is found in health, and is markedly increased in disease of the pyramidal tracts, in heightened irritability of the gray substance of the spinal cord, in many tumors of the brain, in cerebro-spinal sclerosis, in lateral sclerosis, after epileptic seizures or unilateral convulsions, in spinal irritability.[2] It is absent in locomotor ataxia, even at an extremely early age of this affection. It is also abolished in infantile paralysis, in destructive lesions of the lower part of the cord, in neuritis of the lower extremities, in advanced stages of pseudohypertrophic paralysis, and, temporarily at least, as pointed out by Hughlings Jackson, in meningitis and in instances of emphysema and other maladies in which the blood has become venous to an extreme degree.[3] Exceptionally it may be absent in healthy persons; I have known it so in three brothers.

The *tendo-Achillis jerk* is elicited by tapping the tendon when the leg is extended and the foot flexed. If this reflex be exaggerated it may appear when the muscles on the anterior part of the leg or the tibia are struck. This constitutes the *front-tap contraction.*

The *biceps reflex* is developed by tapping the tendon of the biceps. This leads to contraction of the biceps muscle. Its meaning is the same as that of the knee-jerk. Tapping on the front of the wrist gives rise to contraction in the flexors of the fingers; striking the tendon of the *triceps* above the olecranon causes contraction in the triceps. This is especially marked in the irritable muscle of the early and late rigidity of hemiplegia. Another deep reflex is the *periosteal.* It is produced by tapping the bones of the forearm or leg, which gives rise to active contraction of the muscles, and indicates a disease of the spinal cord, especially amyotrophic lateral sclerosis. A slight *jaw-jerk*, elicited by striking the lower jaw obliquely when the mouth is slightly opened, is present in health, and exaggerated in spastic states. Under the latter conditions active flexion of the great toe gives rise to clonus. The *toe-reflex*, described by Sinkler, is met with only when the knee-jerk and ankle clonus are highly developed. The great toe is strongly flexed; immediately involuntary flexion of the foot follows, then of the leg, and of the thigh on the pelvis.

In some instances of disease the reflex phenomena are produced

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[1] Phila. Med. Journ., April 1, 1899.

[2] Hughlings Jackson, Medical Times and Gazette, Feb. 1881.

[3] Brit. Med. Journ., 1892, No. 1614.

on the side opposite to the one acted on. These *crossed* reflexes are not unfrequently met with in posterior spinal sclerosis, and are not merely associated contractions. A tap on the tibia near its middle generally induces contractions of the quadriceps femoris; and it is often followed by contractions of the quadriceps of the opposite leg when both the pyramidal tracts are diseased.[1]

The phenomenon called *reinforcement of a reflex* may have its use and significance in the diagnosis of doubtful or obscure cases. In testing the muscular power of the hand by the dynamometer, it is well known that one hand has greater power if the other hand be forcibly and synchronously clinched. Any reflex is heightened by coincident muscular exertion of other parts than those being tested, and, if a desired reflex be difficult to elicit, it may be brought out by muscular tension of some other part of the body. Strong irritation of the skin acts in the same way. So slight an outlay of force as that of winking will increase the force of the knee-jerk, if correctly timed.[2] When the muscle is cut off from connection with the spinal centres, as in the late stages of locomotor ataxia, the reflex and any reinforcement are alike impossible.

Very similar to the knee phenomenon is the foot phenomenon, or *ankle clonus*, although its reflex character is more doubtful. It is produced if the foot be suddenly brought into complete flexion by the hand pressed against the sole, the leg being semiflexed, and still more readily if subsequently the tendo Achillis be quickly tapped. A kind of convulsive shaking of the foot results, dependent on alternate contraction and relaxation of the anterior tibial and calf muscles. Ankle clonus is at times, but not often, observed in healthy persons, although it is susceptible of being cultivated; in lateral sclerosis it is developed to an extraordinary degree. Indeed, it is marked in the class of affections in which the knee reflex is excessive. When produced solely by sudden passive tension of the muscle, it is indicative of structural change in the spinal cord.[3]

Wrist clonus may be induced in the late rigidity of hemiplegia by pressing the hand backward so as to produce extreme extension at the wrist.

A *muscle-jerk* is obtained by directly striking a muscle, as, for instance, the quadriceps femoris; a contraction ensues. The muscle-jerk may be manifest when the tendon-jerk has ceased; it may be,

[1] Ross, *op. cit.*, vol. i.

[2] Mitchell and Lewis, Tendon- and Muscle-Jerk, Amer. Journ. Med. Sci., vol. xcii., 1886.

[3] Gowers, Diseases of the Nervous System.

indeed, found to be exaggerated. Unlike the tendon-jerk, therefore, it is independent of disease or injury to the motor or sensory nerves of a muscle, or of damage to its related spinal centre.

If a muscle be suddenly relaxed, a slow tonic contraction follows which may last for some minutes. The phenomenon is best witnessed in the tibialis anticus, but is rarely seen in the muscles of the arm. This *pardoxical muscular contraction* has no definitely ascertained value. It is sometimes met with in the early stages of locomotor ataxia.

DERANGED MOTION.

The chief manifestations of deranged motion resolve themselves into the phenomena called paralysis, ataxia, tremor, spasms, and convulsions.

Paralysis.—When we speak of paralysis, we mean a loss of the power of motion, although there is the impulse of the will to move the affected part. It is true, there is also a paralysis of sensation, a complete anæsthesia, which may be conjoined with the paralysis of motion; but the latter often happens alone, and is the morbid state meant when we use the word paralysis without qualifying it. A slight, incomplete paralysis is called "paresis," and this term is especially employed when the loss of power exists without demonstrable organic change.

Paralysis may involve one member, and is then known as *monoplegia*, such as brachial or crural; one-half of the body, *hemiplegia;* both sides of the body, *diplegia;* of the lower extremities, *paraplegia*. When power is lost in the extremities on one side and facial muscles on the other side, the paralysis is designated "alternate" or "crossed."

Palsy may come on rapidly or appear slowly. But under any circumstances it is not a disease, but a symptom. The causes which give rise to paralysis may be thus summed up:

Paralysis due to a lesion or any morbid condition of the nervous centres.—Hemorrhage into or softening of the central nervous textures, or any other process which materially alters or compresses them, or interrupts the main conducting paths, occasions loss of power in the part over which their influence in health extends. The complete paralysis attending most of the diseases of the brain and of the spinal cord belongs, therefore, in this category.

But besides these palsies of organic origin there are *functional palsies*, dependent upon what, so far as we are aware, is simply a functional derangement of the great centres of innervation. Hysterical paralysis, and that occurring after overwork or excesses, and from nervous exhaustion, are examples.

gravis pseudoparalytica, the designation given by Jolly[1] to a peculiar condition characterized by undue readiness of fatigue of voluntary muscles after ordinary functional activity. In fatal cases no distinctive or constant lesion has been found.

Paralysis due to the presence of poisons in the system.—The toxical effects of lead, of arsenic, of mercury, of alcohol, and of sulphuret of carbon, may exhibit themselves by producing palsy. Malarial poison, and poisons formed in the system, such as that of rheumatism or of gout, may act in the same way. The former occasions that singular "intermittent paralysis" which may come on either as one of the phenomena of a fit of ague, or as an apparently independent complaint, which may assume either the quotidian or tertian type, and in which both sensation and motion may be affected. How these poisons operate, whether by interfering with the nutrition of the nervous centres and weakening their generating force, or by enfeebling the conducting power of the nerves, or, as some of them undoubtedly do, by setting up a neuritis, is not fully determined. The palsies coming under this head, being for the most part functional, are not ordinarily intractable. Those due to malaria show the malarial corpuscles in the blood,[2] and yield speedily to decided doses of quinine. Similar to the palsies of poisons and certain cachexias are the palsies after acute diseases. Yet structural changes have been found in these paralyses of supposed blood origin, and many of them are owing to neuritis.

All cases of *periodic* paralysis are not due to malaria; a number of instances have, indeed, been recorded which were not.[3] They are characterized by transitory and recurring muscular weakness of varying degree and distribution, but without other constant or distinctive symptoms. Sometimes there is diminished electric irritability of the affected muscles. There may be also enfeeblement of the reflexes, sensory phenomena, and increased thirst. In some cases, further, a family tendency is present. In a thoroughly studied case of John K. Mitchell's five instances happened in the mother's family. The condition was thought to be autotoxic.[4]

In paralyzed parts the nutrition and secretion are disturbed and the circulation is sluggish; they are frequently swollen and œdematous, the pulse is weaker than in the sound members, and the

[1] Berliner klinische Wochenschrift, 1895, No. 1, p. 1.

[2] See a paper published by me in the International Clinics, 1891, vol. iii.

[3] An elaborate study of this subject has been published by Taylor, Journal of Nervous and Mental Diseases, Sept. and Oct. 1898.

[4] Transactions of the Assoc. of Amer. Phys., 1899.

Paralysis due to a lesion in the course of a nerve.—The nervous force may be properly generated, but the nerve-fibres may be incapable of conducting it. For instance, if a nerve be wounded or compressed, paralysis of the muscles which it supplies takes place. Palsy from this cause is local, and is apt to show marked nutritive changes in the affected part, such as glossy fingers and swollen joints, and to be associated with pain.

Paralysis due to an affection of the nerves at their extremities.—An illustration of such a disorder is the palsy resulting from exposure to cold. Peripheral palsies lead quickly to atrophy of the muscles. They are, from their very nature, local, and commonly remain so. But many peripheral nerves may become implicated, and extensive palsies result, as seen in multiple neuritis.

Motor paralysis due to cold may be met with as a family affection. It has been noticed as thus happening in twenty-two persons, and is clearly described by Rich.[1] On exposure to cold and damp, especially after depressing conditions, the muscles become fixed and immovable in tonic spasm. Respiration, cardiac action, cerebral phenomena, and sensibility are unchanged; the muscles of deglutition are affected if cold substances be swallowed. There is intense desire to urinate, but no derangement of micturition; the sphincters are undisturbed. Motor power returns gradually and progressively on exposure to heat. Recovery is followed by a sense of exhaustion. The disease is hereditary through many generations. It affects both males and females.

Paralysis due to serious interference with the circulation.—This kind of palsy is observed if the principal artery of a part be obliterated. It is sometimes noticed as a transient phenomenon after the ligation of a large artery. If the vascular supply of the brain be interfered with by the occlusion of a vessel, whether by embolism or by thrombosis, the hemiplegia that results is more permanent and very marked. Far advanced arteriosclerosis may also be among the causes of palsy.

Paralysis due to a morbid state of the muscles.—It is doubtful if it be correct to call that paralysis in which the nervous system is not primarily or particularly concerned. Yet certain forms of rheumatic palsy and of muscular atrophy in which the nervous implication is uncertain, but which entail loss of muscular power, may be mentioned here.

A loss of muscular power simulating paralysis is seen in *myasthenia*

[1] Medical News, Aug. 25, 1894.

degree of abolition of muscular motion, carefully contrasting it, when one sided, with the movements of the other side. Is the motion completely abolished or only impaired? what muscles particularly are affected? are concerted movements possible or is there incoördination? and is the gait disturbed? Moreover, what amount of muscular effort is required to overcome special resistance? how is the balancing power? and how are delicate and combined movements executed when the eyesight is withdrawn?

When the power in the arms is only impaired, not lost, we ascertain the degree roughly by the strength of the grasp. But we can do so accurately by a dynamometer. Of these, the best is that of Mathieu (Fig. 13), consisting of a steel ring, slightly elastic, which is pressed firmly in the hand and records the pressure.

The ability of the patient to preserve an erect position, *station*, must be noted as well as the degree of *swaying*, and whether he does so when the feet are brought together and the eyes closed (Romberg's

FIG. 13.

sign), or also when the eyes are open, which bespeaks a much higher degree of disorder. The normal sway with the eyes open is in adults about half an inch forward and backward, and three-quarters of an inch laterally. With the eyes closed it is much greater.

But the most valuable agent by which to judge of the state of the muscles is *electricity*, especially the forms of it known as the induced current, or "faradization," and the constant current, or galvanization," and the action of each must be separately studied. The parts to be examined should be in similar positions. We must begin with a weak current, and the wet electrodes are placed, one on the muscle itself, the other on some other part of the muscle or some indifferent point. This is the *direct excitation* of the muscle. Or the muscular action may be evoked by stimulating the motor nerve supplying the muscle to be tested. This is *indirect excitation;* and in healthy muscles the same strength of current will produce the same amount of contraction whether muscle or motor nerve be stimulated. It is also important to break the current by slow interruptions

especially in employing the galvanic current, to compare the positive (anodal) and the negative (cathodal) opening and closing contractions of the diseased with those of the sound side. In both currents, too, we should ascertain what the quantitative changes are,—whether the muscles react under a feebler current than is usual or require one of great strength to move them. The response depends upon the integrity of both the muscle and the motor nerve. If this be preserved, contraction takes place with every change in a current of sufficient force. With every interruption and with each establishment of the current the muscle can be seen to contract in health, provided the intervals between breaking and making of the current be not too short. The readiness of response to the faradic current is alike, whichever pole is applied to the muscle or nerve, and also when the current is made and broken; but, with the galvanic current, the readiness of reaction varies both with the electrode used and with the making and breaking of the current.

Diminished or lost electro-muscular contractility is a most valuable sign in destructive diseases of the cord. Indeed, speaking in general terms, we may say that it belongs to spinal palsies, while the electro-muscular contractility is intact in cerebral palsies. But this is only true of spinal palsies when the muscles are separated entirely from the influence of the cord: those supplied by nerves having their origin in healthy spinal texture preserve their normal irritability. In fact, if the uninjured part of the cord have become irritated, or more vascular, the muscles having a nervous connection with it may show increased susceptibility to the electric current, and more energetic contraction. We also find diminished electro-muscular contractility when the nerve itself is injured; when there is a mere local change in the muscular texture of the helpless part; and as the result of certain poisons, as of opium, lead, rheumatism, or other blood-poisons, which lower the power of nerve, of muscle, or of nerve-centre. We find it also when there has been long disuse of a limb, as in old cases of hysterical palsy, and even of cerebral palsy. But this is temporary, not permanent; for using the battery for a few days makes the greatest change in the electro-muscular contractility.

As already stated, the electro-muscular contractility is normal in the forms of palsy due to brain disease. The palsied limb may have, indeed, its muscles more powerfully convulsed by a current of the same intensity than those of the sound side are, and then we may infer, as Todd[1] and Althaus[2] have shown, that the paralysis is due to

[1] Clinical Lectures on the Nervous System. [2] Medical Electricity.

brain disease of an irritative character. In recent hemiplegias, whatever their origin, increase of electric excitability is not uncommon. The response of muscle to faradic stimulation is called *faradic excitability;* and the remarks made are based on the effects obtained by faradization.

With reference to the galvanic or continuous current, or *galvanic excitability*, we find that in a healthy state of the muscles the galvanic current will give the same results as faradization, whether muscle itself or its motor nerve be acted on. Healthy muscle and nerve react most readily to galvanic stimulation when the negative pole is applied and the current is made, and, successively, when the positive pole is applied and the current is either broken or made, and, finally, when the negative pole is applied and the current is broken. This so-called "normal formula" may be represented graphically as follows: K.Cl.C.; $\left\{\begin{matrix}\text{An.Cl.C.;}\\ \text{An.O.C.;}\end{matrix}\right.$ K.O.C.; in which K (cathode) stands for the negative pole, An (anode) for the positive pole, Cl (closure) for the making, and O (opening) for the breaking of the current, and C for the muscular contraction. In diseased conditions galvanism may show the same or it may show different reactions from faradism. The muscles of a palsied part may respond actively to galvanization and not at all to faradization. We observe this when the muscular tissue has begun to atrophy and to degenerate in consequence of extensive lesions of the cord, in degenerative affections of the motor roots, in traumatic nerve lesions, and in diseases of the peripheral nerves. While the faradic excitability declines or is lost, the galvanic excitability not only remains, but may be even exaggerated; and in this "reaction of degeneration" (De. R.) there are also complete changes in the normal laws of electric muscular contraction; the anodal closing contraction equals or even exceeds the cathodal closing contraction, the cathodal opening contraction declines in the same manner. There is a deviation from the normal order of response, and thus we note qualitative and not merely quantitative modifications. Again, we may find dissimilarities by interrupting the galvanic current, and these may vary whether the current be rapidly or slowly broken. Thus, Russell Reynolds has shown us that in certain instances of facial palsy from exposure to cold, or in paralysis of the limbs from the same cause, or in lead palsy, the muscles act as little under the rapidly interrupted galvanic current as under faradization; but if the galvanic current be slowly interrupted, they exhibit a greater amount of irritability than do the healthy muscles. In these cases it is found that the muscles are primarily affected, and the application of slowly

interrupted galvanism is rapidly of much service. If is, indeed, well in all cases of palsy, whatever be the form of battery employed, to note the differences in the contraction of the muscles produced by slow or by rapid interruptions. *Static* or Franklinic electricity may also be employed for purposes of diagnosis. We meet with instances where muscles contract under its use which do not respond to either the faradic or the galvanic current.[1]

As already stated, a muscle may be indirectly acted on; one moistened electrode is placed over the motor nerve which controls the muscle, the other over its body. In inflammation of the nerve irritability of the muscle, both galvanic and faradic, is increased; in destructive injuries it lessens and disappears. It is always well to note the indirect as well as the direct muscle excitation. But it has not, for purposes of diagnosis, proved itself as generally valuable. We should endeavor to place the one or other of the sponges exactly over the seat of chief nerve-supply in the muscle; and the ascertainment of the nerve point or points that correspond with the entrance of the motor nerves into the muscles has been made a matter of much study. Experience, indeed, proves that from these motor points, determined with infinite care and labor by Ziemssen, knowledge now accessible in any work on medical electricity, and in most on nervous diseases, the readiest control of the muscles is obtained.

When the muscles react under electricity the contraction is felt, and the "electro-muscular sensibility" is more decided the stronger the contraction. Hence we almost always find increased electro-muscular contractility with increased electro-muscular sensibility. But the latter may exist alone, as we mostly observe in myalgias. On the other hand, the relationship between diminished contractility and sensibility may be changed, as we find, for instance, in the striking want of sensibility to the current in hysterical paralysis. The electric reactions of the skin, well tested by a metallic brush, as a rule go hand in hand with the reactions of the muscles, increase in sensitiveness with them, decrease with them.

Such are the chief facts with reference to the diagnostic applications of electricity in paralysis. There is yet another mode of investigation which we constantly bring into use, one also in which the action of the muscles particularly gives us valuable information concerning the state of the nervous system,—the testing of *the reflex excitability*. But we have already examined into the derangement of

[1] See an excellent summary of the diagnostic value of Franklinic electricity by Bernhardt, Samml. Klin. Vort., No. 41, Feb. 1892.

the reflex system, and shall only add here a few general clinical facts. We find the reflex excitability diminished in disease of the gray substance of the cord, in disease of the sensory root-fibres, which thus become incapable of conducting the impression, and in disease of the motor fibres, which fail to impart the motor impulse. In the latter case there is coexisting paralysis of motion; in the second, anæsthesia. Increase of reflex excitability, producing twitching or even violent irregular movement on very slight stimulation, is found in all irritative lesions which have increased the excitability of the gray substance of the cord; as when this is disturbed by inflammation, or compressed by a tumor, or heightened by certain drugs, such as strychnine. Increase of reflex excitability is also found in parts below a lesion, when this gives rise to descending degeneration in the pyramidal tracts.

As regards the action of the brain, there are instances in which, if all power of appreciating impressions be lost, as in overwhelming cerebral apoplexies, reflex action may be everywhere suspended. On the other hand, irritation transferred from diseased to healthy parts of the brain may produce spasms or palsy phenomena; or the reflex actions may be excited in other parts of the body, as the muscular contractions in the legs during catheterization or in colics. Here the seat of the perverted reflex action is entirely in the reflex areas of the cord.

All these remarks tell us how to examine paralysis. Having now studied the modes in which this is investigated, I shall merely recall that to find out the cause of the difficulty we have to take into account the history of the case, and the attending symptoms, nervous and otherwise; and in eliciting these we should never forget to bring out prominently those shown us by the opthalmoscope, and by examination of the urine and of the heart.

Let us proceed to the clinical study of palsies.

HEMIPLEGIA.

We shall first consider that form which almost always results from brain disease,—hemiplegia, or one-sided palsy. This state of things may affect all the voluntary muscles on one side of the body; but it generally exists only in those of the limbs and face; the eye, neck, and trunk muscles escape largely, though not entirely. Neither the legs nor the arms can move, and the muscles of the face on the side corresponding to the paralyzed limbs are motionless. The cheek hangs; the mouth is drawn towards the healthy side, because the muscles on the other are powerless to resist; the tongue, when protruded, is

ordinarily slowly pushed out towards the palsied side; the articulation is imperfect.

But the rule with respect to the face being paralyzed on the same side as the rest of the body has its exceptions. Should the lesion be seated in the brain, above the crossing of the facial nerves, both face and body are paralyzed on the side opposite to the diseased spot. Should, however, the lesion involve the facial nerve-fibres at a point below or after their decussation, there will be paralysis of the face on one side and the limbs on the other, the facial palsy being direct, and that of the body being crossed.

Now, according to Gubler, this *cross paralysis* is always indicative of a lesion of the pons Varolii, close to which the facial nerves originate, and through which the nerve-fibres for the limbs pass before they decussate lower down. But we must remember that there are rare cases of "alternating hemiplegia," due to a combination of lesions, one affecting a cerebral lobe on one side and the facial nerve on the other. Even when the lesion is unilateral, we may meet with exceptional cases; and, as Bastian[1] points out, the lesion may be situated in the pons, the palsy of face and limbs not being alternate, provided the disease occur in the upper or anterior part of one lateral half, implicating the fibres of the facial above their sites of decussation. With reference to the other cerebral nerves, should we find any of them paralyzed on one side and the body on the other, we shall generally be correct in assuming that the palsy is not due to disease on both sides of the brain, but is rather a disturbance of the affected nerve near its origin or in its course, and on the side on which the brain is injured, while the paralysis of the limbs is on the opposite side. Anatomical researches which have traced connecting nuclei on the floor of the fourth ventricle and elsewhere explain these alternating palsies.

Hemiplegia, as already stated, results, in the vast majority of instances, from cerebral diseases. Hence we find it commonly associated with disordered mental powers, and other signs of a brain lesion. The superficial reflexes are, as a rule, though not invariably, diminished; the deep reflexes are exaggerated. The rectum and the bladder perform their functions. The non-paralyzed side is not wholly free from signs of disorder. Mills[2] has given us an interesting study of its condition, and we see that considerable loss of power and associated movements with any on the paralyzed side are common.

[1] Paralysis from Brain Disease.

[2] The Nervous System and its Diseases, 1898.

Hemiplegia caused by an affection of one-half of the spinal cord, near its beginning, is not combined with a decay of the mental faculties, but the muscles of the chest and abdomen are involved in the paralysis, which they are not in cerebral hemiplegia, unless the lesion be very extensive. Then in *spinal hemiplegia* there is a zone of anæsthesia on a level with the lesion, and coexisting anæsthesia, as Brown-Séquard has shown, on the side opposite to the lesion and the muscular palsy, and the temperature sense is impaired, as is the sensibility to pain; the palsied limb gives evidences of vasomotor paralysis, has at first a higher temperature, and is hyperæsthetic; reflex action is increased on the side of the lesion, the muscular sense is impaired, and the umbilicus is with every act of inspiration drawn towards the sound side. We possess a further test in electricity: unlike what we find in cerebral paralysis, the electro-muscular contractility is greatly lessened or is lost. Spinal hemiplegia, or "hemiparaplegia," as it is more often called if the lesion be low down, occurs from injuries, tumors, syphilitic disease of the cord, and localized sclerosis. Spinal hemiplegia is more persistent in the leg than it is in the arm. In hemiplegia due to cerebral disease recovery is more rapid and more nearly perfect in the leg than in the arm.

But supposing that we have settled the hemiplegia to be cerebral, the points next to be investigated are, where is the lesion situated? and what is its nature? Now, the former question, concerning the *anatomical diagnosis*, may be answered in a general way by stating that the disease is on the side opposite to the palsy, if the lesion, as it almost always is, be seated above the point of decussation of the pyramidal columns of the medulla; for a lesion below the decussation gives rise to palsy on the same side, and a lesion on a level with it, to double-sided palsy. Lesions of the posterior segment of the internal capsule give rise to typical hemiplegia; sometimes with hemianæsthesia and loss of the special senses. Lesions of the corpus striatum cause motor and sensory symptoms only when they involve the internal capsule. The same is true of disease of the optic thalamus, except that mobile spasm and incoördination of movement have been observed to follow lesions of its middle third.

The nearer the lesion to the surface, the more marked are the mental phenomena, the greater is the tendency to spasms in the limbs, but the more limited is the palsy; and the farther the disease extends towards the internal capsule, the more extensive does the paralysis of motion become. We may further distinguish the palsy which ensues from that caused by an affection lower down, as of the *pons Varolii*, by observing that, besides the peculiar crossed paralysis

of the face and limbs, we find giddiness and a tendency to vomit; either loss of the conjugate movement of the eyes towards the side of the lesion, or conjugate spasm with nystagmus; jerkings of the muscles of the face on the side opposite to the injury; sensations of tickling in the face; one-sided facial anæsthesia, with a loss of sense of taste on the corresponding side, though with unimpaired motion of the tongue; rigidity of the limbs, and spasm of the muscles supplied by the fifth nerve; disturbance of respiration and of the heart; albuminuria; glycosuria; high temperature. Should we encounter paralysis of sensibility and motion on one side of the body, and both sides of the face be palsied as to motion and sensation; should the recti muscles of the eye be paralyzed, and taste be lost over the anterior part of the tongue, we may infer that the injury is seated rather above the lower portions of the pons, and affects the spot where the facial nerve and part of the trigeminal cross.[1] Hyperpyrexia is not uncommon after the onset of an acute lesion of the pons, and in acute lesions convulsions[2] are also usual, as is marked contraction of the pupils. In lesions involving the central parts of the pons, paralysis, mostly unequal, of both sides of the body, with impaired sensation, irregular facial palsy, difficulty in deglutition and articulation, is the rule.

Lesions of the lower and inner part of the *crus cerebri* are recognized by an alternate paralysis, in which the third nerve is palsied on the affected side of the brain, showing us want of action of the muscles of the eyeball, except the external rectus and superior oblique, ptosis, a dilated pupil, a tongue deviating to the paralyzed side, some difficulty in articulation, the palsy marked in the arm and leg, and coexisting with increased local temperature, vasomotor disturbance, and very defective sensation.

Acute lesions of the medulla are likely to destroy life; in case they do not, the resulting symptoms are often bilateral and include derangement of the functions of the bulbar nerves.

Besides these well-attested facts, the brilliant researches of the day on the localization of cerebral functions have solved, and are still solving, many problems as important to the physician as to the physiologist. Let us look at some of the additions to pathological knowledge which appear the most certain, premising that in localization only symptoms that are permanent are of value, since any lesion, an acute lesion especially, may for the time being cause vascular or inhibitory disturbance in adjacent parts. We must also be mindful of

[1] Brown-Séquard, Dublin Quarterly Journal, May, 1865.

[2] Gowers, Diseases of the Nervous System.

Broadbent's law, that one-sided movements can be excited from either hemisphere, and that the loss may be soon compensated by the hemisphere with which they are not habitually associated. This becomes often manifest in damages of the cortex.

We shall first glance at lesions of the *motor zone*, including the ascending frontal and parietal convolutions, the anterior two-thirds of the superior parietal lobule and paracentral lobule, parts supplied by branches of the middle cerebral artery. A lesion of these cortical structures causes paralysis of motion without marked loss of sensation. The hemiplegia is more or less complete according to the extent of the motor area involved. It is on the opposite side to that of the disease, and neither the nutrition nor the electric contractility of the palsied muscles is impaired.

The *cortical hemiplegia*, when sudden, is less frequently accompanied by loss of consciousness, is rarely complete from the first, affecting, perhaps, at the onset only the face, an arm, or a leg, and is soon followed by rigidity of the palsied parts. It is apt to be transitory, to show slighter differences in temperature between the two sides, and to be accompanied by localized pain in the head, which may be elicited by percussion over the seat of the lesion, and by temporary aphasia.[1] There is no impairment of sensation in lesions of the motor cortex.[2] Limited palsies, *monoplegias*, are much more common in disease of the cortex than in disease of deeper parts. The leg alone is affected in lesions of the medial cortex or those near to the longitudinal fissure. Irritative lesions of the cortex have as their most characteristic sign unilateral convulsions. In disease of the middle third of the central convolutions the convulsions generally begin in the hand. Disease of the ascending frontal convolution, behind the inferior frontal, gives rise to paralysis of the face, lips, and tongue.

Lesions confined to any one of the *gray central ganglia*, where the internal capsule is not involved, do not afford any special feature by which they may be distinguished from common cerebral hemiplegia. There is paralysis of motion only, which, Charcot[3] tell us, is generally transitory. If the anterior two-thirds of the posterior limb of the *internal capsule* be involved, the palsy is still exclusively of motion, though it is more or less persistent, and ultimately accompanied by muscular contractions; if the posterior third be also involved, we

[1] Ferrier, Localization of Cerebral Disease.

[2] Mills, The Nervous System and its Diseases, 1898.

[3] Lectures on Localization in Diseases of the Brain, New York, 1878.

have in addition cerebral hemianæsthesia. Smell may also be lost on the anæsthetic side, and hemianopsia be met with. In disease of the angle and posterior segment of the internal capsule we have hemiplegia of the ordinary type. Indeed, in lesion of the corpus striatum the hemiplegia is permanent only if the internal capsule be involved in the damage.

A lesion of *one optic tract* or of the cortical visual centre in the occipital lobe will cause bilateral homonymous hemianopsia; a similar effect is sometimes produced by a lesion of the corpora geniculata on one side. There may be considerable hebetude, but no other marked symptom of an affection of the brain except hemianopsia. In lesions, also, of the *prœfrontal lobes*, that part which, in its relation to the skull, is roughly bounded by the coronal suture, there is no disorder either of motility or of sensibility. The manifestations are simply those of restlessness and unsteadiness of mind, mental apathy, impairment of judgment and reason, and other psychical disturbances; a tendency to make jokes has also been noted.[1] Yet the frontal lobes of one side may be totally destroyed without changes in mind or character.[2] There is no motor paralysis except of the foot. Late in the case, among pressure and invasion symptoms, we may find motor aphasia, nystagmus, and unilateral convulsions.[3] In disease of the *temporo-sphenoidal* lobe we have deafness in the ear opposite to the lesion, if left-sided sensory aphasia, and sometimes convulsions with preceding auditory aura. There is no hemiplegia.

The *nature* of the paralyzing lesion, the *pathological diagnosis*, can be arrived at only by a careful scrutiny of all the facts of the case. A sudden paralysis occurring simultaneously with coma almost always has its origin in an apoplectic effusion, more rarely in cerebral embolism or thrombosis. A sudden paralysis without coma is generally due to plugging of the vessels. A gradual development of palsy indicates some chronic cerebral disease, such as chronic endarteritis with altered brain nutrition, or a tumor, or any affection compressing the nervous substance. We may also gain much knowledge by carefully exploring the organs of circulation and the kidneys. Thus, a paralysis found to be conjoined to a cardiac malady or to a diseased state of the arteries is, in all likelihood, owing to a clogging of one of the cerebral arteries, and to consequent tissue-change in the cerebral structures. When the kidneys are seriously disordered, the

[1] Oppenheim, "Geschwülste des Nervensystem," in Nothnagel's System.

[2] Case of Bailey, "Hemiatrophy of the Brain," Amer. Journ. Med. Sci., March, 1899.

[3] Mills, Cerebral Localization in its Practical Relations, 1889.

hemiplegia is likely to be caused by some chronic disease of the brain or its vessels, the result of an altered nutrition. The uræmic condition itself seems also capable of causing loss of power, sometimes of hemiplegic type.

In paralyzed limbs we are apt to meet with rigid states of the muscles due to tonic spasm, which, when they produce spastic muscular shortening, are called contractures. Under ether or chloroform anæsthesia these disappear. When the paralyzed limbs exhibit a rigid state from the moment of or soon after the attack, the *early rigidity* points to an irritative lesion, such as a compression of healthy brain-tissue by an apoplectic clot. *Late rigidity*, if persistent, generally becomes associated with wasting of the muscles, and with central degeneration of the motor tracts. It is generally combined with excessive tendon reflexes, muscle jerk, and with ankle clonus. Under excitement the paralyzed arm and leg may be strongly flexed, and automatic movements may occur when the patient sneezes.[1] We may also on the palsied side meet with tremors; with attacks of true spasms, happening particularly in the arms; with joint-disease and nodes; and with choreic movements, a condition to which, under the name of "post-paralytic chorea," Weir Mitchell[2] has called attention. In some cases of hemiplegia there is much pain in the stricken limb. The pain may precede returning motion, and is thus of favorable augury. But in limited disease of the internal capsule affecting the sensory path the pain in the palsied limbs may persist through life. In old hemiplegias the surface temperature is lower than on the non-paralyzed side.

Hemiplegia may be *feigned*.[3] But the results of electricity, especially where altered sensibility as well as defective motion is simulated, and the test proposed by Hughlings Jackson, that the arms do not, as in real hemiplegia, fall forward when the patient stoops, but are retained at the side, will usually detect the fraud.

MONOPLEGIA.

When we have limited lesions we have limited palsies, and researches on localization are teaching us more and more accurately to recognize the centres affected in these palsies of special parts, or of one limb, or of a group of movements. Of course, in making a diagnosis of the paralysis being due to disturbance of a special nerve-

[1] Ross, Diseases of the Nervous System, 1883, vol. i. p. 187.

[2] Amer. Journ. Med. Sci., Oct. 1874; also Med. News, April, 1893.

[3] For an instructive case see London Lancet, April, 1874.

centre, we must be careful to exclude, as the cause of the local palsy, peripheral affections, and those in the course of the nerve supplying the stricken part, and also to make it clear that the lesion is not spinal of very circumscribed kind. In monoplegias the palsy is never complete. Furthermore, it is always important in a given case to separate the symptoms which may be due to invasion of or to pressure on adjacent centres from the localizing symptoms of the main lesion. Let us now take up some of the limited palsies dependent on cerebral disease, especially in the motor areas of the cortex.

One arm only is paralyzed.—Here we find the lesion in the ascending parietal and the ascending frontal convolution on the side opposite to the palsy, and the disease is limited to the middle third of the convolutions. If the lesion be double, as in a case referred to by Bourdon,[1] both arms are helpless. But, whether single or double, with the damaged motion there are unimpaired sensation and electro-motor contractility. Disease of the ascending frontal opposite the upper half of the inferior frontal convolution gives rise to palsy of the lower part of the face except the lips.

One arm and the same side of the face are paralyzed.—In this "brachio-facial monoplegia" the lesion is in the central region of the cortex, towards the middle or lower third of the ascending convolutions in the facial and arm centres. It is a purely motor palsy, associated, however, usually with aphasia when the disease is left-sided. The main movements of the muscles of the upper part of the arm are kept, while those of the hand are lost. Palsy, of cerebral origin, limited to *one side of the face*, without the arm being implicated, is rare; the cortical disease is in the centre for the facial region. The affection is usually left-sided, and is apt to become complicated with aphasia. The lower part of the face bears the brunt of the palsy; unlike Bell's palsy, the orbicularis and the upper part of the face are but little, if at all, disturbed;[2] further, there is no disease of the temporal bone to explain the localized palsy by an injury to the facial nerve. The tongue is also very generally implicated.

The leg only is paralyzed.—This is a very rare form of paralysis, and presupposes a lesion limited to the motor centre for the leg. The centre for the leg and foot is fixed by the researches of Horsley and Schaefer as occupying the uppermost portion of the ascending frontal and parietal convolutions. In some of these cases of "crural

[1] Bull. Soc. Anat., 1874.

[2] This is strikingly illustrated in a case reported by Guitéras, Phila. Med. Times, Nov. 1878.

monoplegia" on record the ascending parietal and superior parietal convolutions have been found diseased. Sensation is not affected; the arm is apt to become gradually involved in the palsy; in Ferrier's case[1] the lesion was in the quadrilateral lobule on the internal aspect of the hemisphere and in the upper extremity of the ascending parietal and frontal convolutions.

There are many other kinds of limited palsies of cerebral origin, such as of the tongue, *glossoplegia*, of the face and tongue, *facio-lingual monoplegia*, of the eye muscles, *oculomotor monoplegia*, and half blindness, *hemianopsia*, to all of which I can only refer, since our knowledge is not definite enough to lay down concise conclusions for diagnosis. In part, too, they will be discussed farther on. It must, however, be added that in all these limited palsies traceable to disease of the brain we are apt to have such symptoms as are common in brain affection,—headache, giddiness, and the like. These aid us in understanding the nature of the disorder.

PARAPLEGIA.

This differs from hemiplegia in the palsy occurring on both sides, yet being, in the vast majority of instances, limited to the lower or the upper extremities. Its almost invariable cause is a lesion of the spinal cord. In truth, if we call hemiplegia paralysis from brain disease, we may call paraplegia paralysis from spinal disease. Paraplegia is generally due to a marked organic lesion; but there are cases in which it exists independently of any recognizable structural change, and in which it results from poisons, from fatigue, from excesses.

The disorder generally comes on slowly. At first the patient only loses the steadiness of his gait; gradually he is deprived of all power of motion, but the intellect and the nerves of special sense remain unaffected. If the lesion be in the lumbar part of the cord, the palsy is confined to the lower extremities and to the pelvic muscles; if the dorsal portion be attacked, we find, in addition, signs of paralysis of the abdominal walls and of the sphincters, tympanites, and somewhat impeded breathing. In disease of the upper section of the cord there is coexisting palsy of the upper extremities, with dilated, sluggish pupils, and difficulty in deglutition and in respiration. In the muscles supplied by nerves which originate in healthy marrow, involuntary contractions or reflex phenomena can be induced,—are, indeed, generally exaggerated,—and the striking effects of strychnine, when given in doses sufficient to produce its peculiar muscular spasms, are mani-

[1] Brain, vol. iii., 1880.

fested. The palsied muscles, in the majority of the affections occasioning the paraplegia, undergo wasting, and often do not respond to the electrical stimulus.

Paraplegia is generally more marked on one side than on the other, and the paralysis of motion is apt to be associated with complete anæsthesia. When, as sometimes happens, the mischief is limited to a lateral segment of any part of the cord, there is paralysis of motion on the same side of the body, and of sensation on the other. Preceding, or even attending, many cases of paraplegia, is a symptom which belongs exclusively to affections of the cord: a spasm of the flexor muscles of the lower limbs, so powerful that the anterior parts of the thighs come almost in contact with the abdomen, while the heels are drawn up so as to touch the back of the thighs.[1]

Let us now take a cursory view of the different forms of spinal paraplegia.

SUDDEN PARAPLEGIA.

Spinal Hemorrhage.—Sometimes the paralysis occurs suddenly, and in consequence of an injury to the spine, of a displacement subsequent to a disease of the bones, of blood extravasated into the canal, of poisons, as the lathyrus sativus,[2] or of bulbar or spinal disorder from sudden displacement of the cerebro-spinal fluid following blows on the head.[3] When either of the first two causes has led to the sudden palsy, the diagnosis is materially aided by the history of the case, and by a close examination of the vertebral column. But if there be no signs of a disease of the bones or of the intervertebral cartilages, we may suspect a *spinal hemorrhage* to have produced the sudden and complete paraplegia, developing as it does in a few minutes; and this suspicion becomes much strengthened if violent localized pain in the back exist or have preceded the rapid palsy, if the patient be unable to retain his urine or fæces, and if the affected limbs be relaxed and largely deprived of sensation. The seat of pain corresponds to the seat of the apoplexy. The pain occurs in distressing paroxysms and passes along the course of the nerves compressed by the extravasation. Where the hemorrhage is meningeal, there is more persistent pain, with rigidity of the spine, spasms of the legs, slighter disturbance of sensibility, much less quickly increasing paralysis, and there is more apt to be spasmodic retention of

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[1] Brown-Séquard's Lectures on the Nervous Centres, p. 114.

[2] Irving, Indian Annals, No. 12, referred to in Brit. and For. Med.-Chir. Rev., Oct. 1860.

[3] Duret, Traumatismes cérébraux, Paris, 1878.

urine. The absence of early fever distinguishes the spinal hemorrhage from spinal meningitis; subsequent fever bespeaks the occurrence of this as a complication. The muscular spasm is sometimes so severe that it has been mistaken for tetanus, which lacks the violent pain in the back. The most common causes of spinal hemorrhage are blows and falls on the back or falls on the feet. It is also met with in diseases with hemorrhagic tendencies, in convulsive affections, and in the course of myelitis. Hemorrhage into the membranes may result from the rupture of an aneurism of a vertebral artery.

Paraplegia sometimes develops in persons who emerge directly from compressed air into the ordinary atmosphere, as, for instance, divers and workers in caissons. There may be besides numbness and tingling, nausea and vomiting, headache, vertigo, a sense of throbbing, palpitation, oppression of the chest, bleeding from the nose, and loss of consciousness. The condition is believed to be due to sudden setting free of gases dissolved in the blood as a result of the increased pressure.

But besides these causes, others lead rapidly to paraplegia. Softening of the cord may have progressed latently until the degeneration destroys the continuity of the conducting tubules, when palsy at once takes place. Then there are cases following violent exercise or sexual excesses, cases for which neither during life nor after death an organic cause can be assigned,[1] and which are regarded as due to enfeeblement of functional power. The disorder is much more apt to come on quickly than gradually, and rest and a tonic treatment are likely to be followed by decidedly good effects. But in regard to all these cases of functional palsy, the same as in regard to reflex palsies, science is more and more narrowing their number by finding some organic affection in the cord, often secondary to an ascending neuritis. Indeed, their very existence is now for the most part denied.

Acute Ascending Paralysis.—Yet another variety of paraplegia which may happen rapidly is that form which has been described as *acute ascending paralysis*, or Landry's paralysis. It may come on after fatigue and exposure in persons in perfect health, generally in men between twenty and forty years of age. Usually there is little or no fever except at the onset. Numbness and tingling, and slight pain in the lower extremities, are soon followed by loss of muscular power, which, in turn, goes on rapidly, generally in a few days, to complete

[1] For instance, Case XVIII. in Gull's series in Guy's Hosp. Rep., vol. iv., 3d Series.

paraplegia. The legs are relaxed and immovable, the muscles of the trunk are next affected, then the upper extremities become implicated, and sensation, which at first was normal, is somewhat enfeebled though never to a marked degree; occasionally the arms are involved before the legs. The patient is restless, sleepless, but his intelligence is, as a rule, unimpaired, and we find no bedsores and no palsy of the bladder or rectum. The respiration and circulation in the progress of the disease become embarrassed, inability to swallow occurs, there is acute enlargement of the spleen, and sudden death ensues within a month from the time of the seizure,[1] or, indeed, the case may end fatally in less than a week. But all cases do not run so rapid a course; and, in truth, we meet with instances in which the disorder is rather chronic than acute, or is arrested. The muscles do not atrophy, and their electrical excitability is unimpaired, which is a very valuable diagnostic test. About the reflexes the statements are conflicting. It is most likely that at first both the superficial and the deep reflexes are absent, and that they do not return, certainly the knee-jerk does not, except when the paralysis passes away. Jaccoud[2] tells us that in the cases he observed the reflex movements were abolished. In Mills's case[3] which recovered, both the superficial and deep reflexes were completely lost. The disease occurs generally between the ages of twenty and forty, and follows toxæmias and infections, such as influenza, diphtheria, typhoid fever, and smallpox. Gowers mentions a case following pelvic cellulitis.[4] The malady is looked upon as being an affection of the peripheral nerves, though the central nervous system is not infrequently involved. Toxic influences of invading micro-organisms are thought to give rise to it.[5]

The disease which most resembles acute ascending paralysis is acute progressive or multiple neuritis. But here sensation is rapidly lost, and so is the electrical excitability.

Multiple Neuritis.—When nerve after nerve rapidly inflames, or the inflammation occurs at one time, an extensive palsy is quickly developed. The disease is an affection of the peripheral nerves, though it has the misleading symptoms of a spinal malady. It attacks both sexes, is most common between the ages of thirty and fifty, and, though it may follow altered blood-states or rheumatism, or be due

[1] As in the case reported by Hayem, Travaux de la Société Médicale d'Observation, tome ii., 1867; see also Leyden's Klinik der Rückenmarkskrankheiten.

[2] Clinique Médicale.

[3] Transactions of the Assoc. of Amer. Phys., vol vii., 1892.

[4] Diseases of the Nervous System, 3d ed., 1899.

[5] Cramer, Centralblatt für Pathologie, Jan. 1892.

to exposure, by far its most frequent cause is chronic alcholism. It has been observed in the sequence of a number of infectious diseases, toxæmias and septicæmias, and also as a result of the medicinal administration of arsenic, of lead, and of silver. It has generally an acute or a subacute beginning, with decided increase in temperature. At first vague, then more decided pains are felt in the extremities, chiefly in the fingers and toes, and these pains soon become darting or burning and may occur in paroxysms. The pain is often preceded by tingling and by cramps, is increased by motion, and is associated with tenderness of the affected nerve-trunks and with both skin and muscle tenderness of the parts to which they are distributed; finally this increased sensibility may give way to anæsthesia.

The palsy shows itself often first in the arms, the earliest loss of power being evident in the extensors of both sides. Soon the muscular weakness is seen also in the legs, and the trunk muscles and face muscles may become involved. But the first signs of palsy may be in the legs, and manifest itself in a peculiar gait. The symmetrical character of the palsy is always noticeable, as are also the double wrist-drop and foot-drop. The parts affected waste, and lose their reflex excitability; the loss of the knee-jerk is especially pronounced. The muscles do not react to faradization, though they may to galvanism; often, indeed, they present the reaction of degeneration: the nerves are uninfluenced by the electric stimulus. Œdema of the arms and legs is frequent, and profuse sweating is not uncommon. Sometimes muscular incoördination is the most prominent symptom.

The disease may run on to complete palsy of the limbs in less than two weeks, and death result from paralysis of the respiratory muscles; or the affection may pass into a chronic condition, and a slow improvement, with return of power in the muscles, take place. In protracted cases contraction of unopposed muscles occasions deformities, and there are arthritic adhesions, glossy skin, and thickening of the skin.

The diagnosis is generally easy. The tingling in the extremities, the cutaneous and muscular sensitiveness, the distribution of the symptoms, the early development of muscular weakness, and the palsy of the extensors distinguish the disease from *rheumatism*. In some instances, where it is difficult to elicit tenderness of nerve-trunks, or where this symptom is wanting, where the muscular tenderness is not marked, where, moreover, the palsy is slight and incoördination of movement is observed, the similarity to *locomotor ataxia* is great, and the eye-symptoms of this affection alone, if present, will help to a correct conclusion. Further, girdle-sense and lightning-pains are

absent in peripheral neuritis, while wasting is not generally observed in posterior sclerosis. In ordinary cases the greatest resemblance is to those instances of *acute myelitis* which run a rapid course, and especially those in which muscular wasting is marked. To *acute ascending paralysis* intense cases of the disease also bear a strong likeness.

In the following table are contrasted the features of multiple neuritis, of acute myelitis, and of acute ascending paralysis.

Multiple Neuritis.	Acute Myelitis.	Acute Ascending Paralysis.
Fever, with at first decided elevation of temperature.	Fever generally moderate.	No, or only slight, elevation of temperature.
Palsy begins in forearms, extends to legs and trunk, or may show itself first in the legs; double wrist-drop and foot-drop; palsies symmetrical.	Palsy generally affects only legs and lower part of trunk, though it may affect arms.	Paralysis rapidly extending from lower extremities; relaxed muscles.
Muscles atrophy rapidly. Trophic changes in skin and nails common; no bedsores.	Muscles atrophy rapidly. Trophic changes marked; bedsores.	No muscular atrophy. No trophic changes; no bedsores.
Marked pain and sensory disturbances, hyperæsthesia especially, later anæsthesia in the area of distribution of the inflamed nerves; muscular tenderness; tenderness of nerve-trunks.	No pain or tenderness of nerve-trunks; complete anæsthesia below lesion; zones of hyperæsthesia corresponding to lesion.	No marked pain or more than dulling of sensation in affected parts; no tenderness of nerve-trunks.
Loss of electrical excitability. Generally reaction of degeneration.	Loss of electrical excitability. Generally reaction of degeneration.	No change in electrical excitability.
Reflex action lost, especially deep reflexes and muscle reflex.	Excessive reflex action, except in parts deriving nerve-supply from injured centres, there lost.	Absence of reflexes the rule.
Sphincters unaffected.	Sphincters affected early.	Sphincters nearly always escape.
No bulbar symptoms, though respiratory palsy may happen.	Bulbar symptoms rare; failure of respiratory power may happen.	Bulbar symptoms frequent.
Mind unaffected, except in the alcoholic and toxæmic cases.	Mind unaffected.	Mind remains clear.

The various causes of multiple neuritis give rise to some differences in the symptoms, by the close study of which we may infer the cause. Thus, in neuritis from metallic poisons the disorder is confined to the arms, as in lead poisoning, or is found in the arms first and subsequently attacks the legs, as in the neuritis of arsenical poisoning. In malarial neuritis the legs are first attacked, and the neural malady may be confined to them. The neuritis due to diphtheria often gives rise to paralysis of the palate, of the fauces, of accommodation, and of the lower extremities, at times closely simulating locomotor ataxia. Neuralgic pain of irregular distribution, with sugar in the urine, is characteristic of the neuritis of diabetes. In alcoholic neuritis all the limbs are affected, the pains are very severe, and both sides of the face may become involved. Symptoms much the same with reference to the distribution of the motor and sensory disturbance happen in the multiple neuritis from cold. In the toxic cases the face is also apt to be involved, and the optic nerves seem to be only in them affected. The symptoms due to the neuritis of leprosy resemble greatly those of syringo-myelia. In pyæmic and septicæmic cases there are the history and the pyrexia to guide us. In the multiple neuritis of influenza comparatively little pain occurs: but considerable palsy, some facial paralysis, and difficulty of swallowing are not uncommon. Multiple neuritis may occur in the old without obvious cause. In some cases it manifests a peculiar tendency to recurrence.

Infectious Paralyses.—These are specially seen in children, and are mostly of the spinal or peripheral type, though they may be cerebral, or occur in varied combination. We may find them affecting the two arms or the two legs, or all four limbs. Many of the cases are clearly instances of multiple neuritis, others of neuritis associated with myelitis: the palsy is frequently very wide-spread. These infectious paralyses are noticed after influenza, typhoid fever, measles, scarlet fever, diphtheria, mumps, whooping-cough. They present clinically the traits of the pathological lesions occasioning them, and in their diagnosis the history of the preceding attack is of the utmost importance.

SPINAL PARAPLEGIA.

This occurs in congestion, in acute and chronic inflammation of the meninges, in myelitis, in softening, in atrophy, in sclerosis, in compression of the cord, and from reflex irritation. These are some of the marks of discrimination:

Spinal Congestion.—There are no certain symptoms of spinal congestion. It is likely that the aching in the spine and legs that

comes on in weakly persons who have remained for a length of time on their backs is due to passive spinal congestion. More active spinal congestion may occur in diseases of the heart and in gouty persons, and is perhaps the cause of the nocturnal muscular jerkings from which they suffer.

Spinal Anæmia.—Except as part of a general anæmic state, or after severe hemorrhages, there is no proof that spinal anæmia exists. The symptoms of hysterical or "irritable spine," or "spinal irritation," a disease met with in hysterical and anæmic young women, are supposed to be due to it. These, besides palpitation, neuralgic pains in the chest and abdomen, and aching in the inactive and sensitive limbs, are pain along the spine, and marked tenderness on pressure on the spinous processes of the vertebræ. But that spinal anæmia is the determining cause is very doubtful. In anæmia of the cord following hemorrhage, which has generally happened from an abdominal organ, the weakness of the legs and arms may pass into complete motor paraplegia. In some instances Leyden has observed attending hyperæsthesia.

Spinal Meningitis.—In inflammation of the meninges we encounter severe pain in the back, little influenced by pressure upon the spine, yet aggravated by movement, even by the acts of defecation and urination; sometimes a sensation as if a cord had been drawn around the belly; pains in the limbs similar to those of rheumatism; cutaneous hyperæsthesia or anæsthesia; muscular twitchings, rigidity, and contractions, more or less permanent and painful; increased superficial and deep reflexes, when the disease is above the lumbar enlargement of the cord; very commonly distressing spasms in the muscles of the back, and spasms in the limbs occasioned by attempts to move them; rigidity of the spinal column; bedsores; dyspnœa; retention of urine; vasomotor derangement; yet only incomplete paralysis, or, indeed, none at all. In the acute form we have decided fever. When marked paraplegia follows the symptoms mentioned, we may suspect the development of myelitis, or that an effusion has taken place which compresses the spinal cord. Cases of spinal meningitis occur from falls and shocks, from exposure to cold, as a consequence of adjacent disease, and in the course of general infectious processes. They are not unusual among soldiers who have slept on damp ground.

As regards the special membranes involved, there is no certainty in diagnosis. The symptoms, save in the acute purulent forms of the disease, are slow in developing. In inflammation of the dura mater, *pachymeningitis*, the radiating pains are very severe, but there is less

vertebral pain and stiffness in the back: these signs are seen in their fullest expression in inflammation of the pia mater and arachnoid. In inflammation of the inner surface of the dura mater, *pachymeningitis spinalis interna*, which particularly happens in the cervical region, the symptoms are chiefly referred thither; and stiffness of the neck, paralysis in the upper extremities, especially in the parts supplied by the median and ulnar nerves, claw-like hands, contractions, severe pains in the arms, spots of anæsthesia, and herpetic eruptions are common. At a later period, as the hypertrophic thickening of the dura extends and the cord is more and more compressed, the paralyzed muscles may undergo wasting. There is a hemorrhagic form of pachymeningitis interna having the same causes as hæmatoma of the dura mater of the brain, and often accompanying it.

Myelitis.—Myelitis presents many of the same symptoms as spinal meningitis, with which, in fact, it may be associated. Frequently the symptoms come on by slow degrees, and the paraplegia, a very distinctive symptom, gradually becomes complete. There is strong knee-jerk with ankle clonus. Contractions of the muscles are uncommon, and not permanent, unless late in the disease; the muscles are usually flaccid; there is comparatively little pain, none on pressure at any part of the spine, or on motion, and anæsthesia sooner or later shows itself. Further, we generally, though not constantly, find the urine alkaline, and, as a rule, retention of urine and a want of control over the rectum exist, bedsores form readily, and the temperature of the palsied is lower than that of the healthy parts.

In acute cases there are, as in acute spinal meningitis, raised temperature and a frequent pulse. The fever is moderate and irregular. There is pain in the back, not increased by movements, and pain in the limbs preceded by numbness or burning. In many instances we notice erection of the penis. Spasm in the extensor muscles is always of significance. Reflex movements in the relaxed palsied limbs are gradually abolished as the process of inflammation and softening affects the gray matter of the cord. In dorsal myelitis the trunk reflexes are impaired, but the reflex excitability remains excessive in the parts supplied by nerves arising below the level of the greatly diseased centres. In disease of the lumbar enlargement the knee-jerks are wholly lost.

An altered sensibility to heat and cold, when, for instance, a sponge soaked in warm water or a piece of ice is applied to the spine over the inflamed spot, has been spoken of as a diagnostic test; in either case the sensation, when the diseased part is reached, changes to a burning sensation. This symptom is, however, far from constant, and cannot

be accepted as conclusive. There is a zone of hyperæsthesia at the level of the lesion, and corresponding to this a zone of constriction or "girdle pain." Below the level of the lesion the loss of sensation is complete. The paraplegia, even in acute cases, is not suddenly developed. Yet we meet with exceptions. There are instances in which it comes on almost as rapidly as in spinal hemorrhage. These are mostly instances of *hemorrhagic myelitis*; yet even these are generally preceded by tingling in the limbs and other sensory disturbance, and there is fever, but not the acute spinal pain of hemorrhage. A paralysis of the bladder may be the first symptom of myelitis, and paralysis of motion and of sensation quickly follow.[1]

Myelitis may be the result of cold and exposure, of over-exertion, of syphilis, of peripheral irritation, of pressure, as from disease of the vertebræ, of tumors, connected with the bones or membranes, of parasites, of aneurisms, encroaching on the cord and setting up disease there, of injuries to the cord, or of concussion of the spine after railway accidents. It is sometimes met with in the course of measles and of smallpox, and of typhoid and typhus fevers and toxic blood-states, such as gout and syphilis. *Compression* as a cause has been noted in the cervical as well as in the other portions of the spine. Paralysis of the arms, with dilated or contracted pupil and very slow pulse, is among the chief symptoms of the "cervical paraplegia." Pain in the limbs, hyperæsthesia, muscular contraction, spasms, and great reflex irritability are among the earlier symptoms of this as of all the other forms of myelitis from pressure; but as the case progresses the reflex irritability is lost. Yet recovery, almost complete, is possible.[2]

Unilateral flushing and sweating have been observed and a retro-pharyngeal abscess may form. When the dorsal region is involved obvious deformity may be present. The paralysis of the lower extremities develops late and progresses slowly. Radiating pains are present, and the knee-jerks, occasionally absent at first, finally become exaggerated. Compression of the lumbar cord is attended with loss of control of the sphincters, while the knee-jerks are lost.

In looking at the symptoms which mark the extent and exact site of the inflammation, we find in the common form, where the disease affects a considerable portion of the thickness of the cord, —*transverse myelitis*,—with the ordinary symptoms of complete paraplegia and anæsthesia, that the reflex excitability is lost in the parts

[1] Erb, in Ziemssen's Cyclopædia, vol. xiii.

[2] Buzzard, Brain, April, 1880.

supplied by the nerves coming from the affected portion of the cord, and is preserved or increased in the parts supplied by nerves arising from the cord below the diseased area,[1] and the muscles respond to the electric current. This is not the case in *central myelitis*, which, moreover, usually runs a rapid course, in which there is speedy loss of sensation and of reflex action, and in which muscular atrophy soon shows itself. In *disseminated myelitis*, a form where several foci of inflammation are present, there are lulls and exacerbations, the paralysis is not so constant nor so complete, although it may be in all four limbs, spastic symptoms are not uncommon, and the disease develops itself after acute maladies, as after smallpox. *Hemorrhagic myelitis* is usually central; the paraplegia comes on in less than an hour. In children the anterior cornua are apt to be affected, and the disease is known as *poliomyelitis*.

Spinal Scleroses.—Sclerosis of the spinal cord may be primary or secondary. The latter is represented by the descending or ascending degenerations that follow lesions of brain, cord, or posterior nerve-roots. In the former are included the so-called system diseases,—posterior sclerosis, locomotor ataxia, and lateral sclerosis. The sclerosis where brain and cord both suffer, we shall discuss with the forms of tremor; posterior sclerosis of the cord produces the symptoms of locomotor ataxia, not of palsy.

Lateral Sclerosis.—Primary sclerosis of the lateral columns in which the anterior horns are not affected shows the group of symptoms described as *spasmodic dorsal tabes* by Charcot, or *spastic spinal paralysis* by Erb. It is characterized by a sensation of weakness in the back, a gradually increasing loss of muscular power in the lower extremities, proceeding slowly from below upward, and associated with reflex spasms and persistent muscular contractions, with increased tendon reflex, but without impairment of sensibility, or trophic disturbances, or bedsores, or vesical disorder. The muscles are well nourished, or only very slightly wasted; the gait is peculiar, the walk being on the toes, and as the foot touches the ground a trembling happens. Sometimes there is marked contraction of the adductors of the thighs, and the knees are in contact or even crossed. The function of the sphincters may be enfeebled or lost. No cerebral symptoms whatever exist; the electrical excitability is either normal or somewhat lessened. In rare instances the disease begins in the upper extremities; it is almost always of very slow

[1] According to Bastian, total transverse lesion high up in the cord abolishes the knee-jerk.

development. Occasionally it terminates in recovery. It is most likely that the disease consists essentially in a primary sclerosis of the pyramidal tracts, or of the terminations of their fibres in the motor cells of the gray matter. But whether the group of symptoms may not be produced by various lesions of the cord is not settled. To an infantile form of degeneration of the lateral columns McLane Hamilton has called attention. Loss of power in the lower extremities, muscular contractions without marked atrophy or greatly impaired electro-muscular contractility, such as happen in infantile paralysis, increased skin and tendon reflexes, and absence of sensory disturbances or brain-symptoms, are the chief signs of the affection.[1]

When sclerosis affects the lateral columns, and is combined with degeneration of the great ganglion cells in the anterior horns of gray matter of the cord, the portion which has a controlling influence over nutrition, marked nutritive changes happen in the palsied part, such as we find in progressive muscular atrophy. But this *amyotrophic lateral sclerosis*, as Charcot has termed it, is from the onset an atrophy of a whole muscular group. It is a disease closely allied to progressive muscular atrophy, and, beginning in the arms, affects as a rule, the four limbs successively, produces strange deformities in the wasted and palsied limbs, that are agitated by fibrillar movements, extends to the hypoglossal and the pneumogastric nerves, and thus determines death.

Tumors of the Cord.—Tumors of the spinal cord, either growing from it or its membranes, or originating in the vertebræ and compressing the nerve-structure, occasion paraplegia. The symptoms vary with the situation and extent of the growth. They depend first upon the irritation, and later upon the compression, caused by the new formation. We suspect the affection if we have signs of a grave constitutional malady attending the slowly progressing palsy, if this be more decided on one side than on the other, and if anæsthesia be found on the side opposite to that in which the palsy is marked. The severe pain over the locality of the disease, at first neuralgic, then becoming constant, is aggravated in paroxysms. The pain is generally felt on one side first, and is associated with tenderness and rigidity of the spine, and muscular spasm or rigidity in the limbs. Yet, unless we have distinct evidence of tumors elsewhere, the diagnosis is never more than an uncertain one. If multiple tumors exist, it may be made positive. Strong proofs of syphilitic infection point to the spinal symptoms being due to a syphilitic growth; and signs of scrofula or tubercle in the lungs or in other internal organs, make

[1] Transactions of the American Medical Association, 1879.

ll likely that similar morbid products are the cause of the palsy. Should a gradually progressing paralysis suddenly show symptoms of acute myelitis in a person with the constitutional cachexia just mentioned, we have an additional reason for supposing the affection to be tubercular and to be rapidly extending.[1] Lymphadenomas elsewhere make it extremely probable that the spinal symptoms are owing to one or several of them in the cord. Yet the spinal symptoms in the affection may be really due to myelitis. In all cases of suspected tumor we must be careful to ascertain that bone-disease is not the cause of the symptoms. The absence of sharp pain and the uniformity of the palsy on both sides are points of distinction as against tumors. The early signs of tumor of the cord suggest hysteria.

Reflex Paraplegia.—Functional disturbance of the cord from irritation causing an inhibition of spinal centres is supposed to give rise to the so-called reflex palsies. Worms in the intestines may occasion them. But the most marked of them is the paraplegia consequent upon disease of the bladder. Yet it is very doubtful whether there is not always in these reflex paralyses organic disease, especially an ascending neuritis, and it is unlikely that reflex palsies have any real existence.

So much for paraplegia. We shall now examine some of the other clinical varieties of paralysis; beginning with a group in which the palsy is limited, though it may be general.

PALSIES USUALLY LIMITED, THOUGH THEY MAY BE GENERAL.

Hysterical Paralysis.—We distinguish this form of paralysis from that of organic disease by its occurrence in hysterical persons; its sudden appearance, and frequently its just as sudden disappearance; its coming on generally under the influence of some powerful emotion, often after an attack of hysterical convulsions; the absence of any signs of a serious lesion of the nervous centres, except the paralysis, the varying nature of the palsy, sometimes hemiplegia, sometimes paraplegia, its incomplete character, the patient being not infrequently able to move while under strong excitement; and the ease with which reflex movements are brought on in the seemingly [illegible]. There are nervous shootings, noises in the ears, [illegible] and vaso-motor disturbances showing [illegible] and elsewhere. The mus[illegible] perfectly under both [illegible] The electro-muscular sensi-

[1] See Lees's Report, Lancet, 1875, and Erb, in Ziemssen's Cyclopædia.

bility is either diminished or abolished. In some cases galvanic sensibility is lost.[1] We never find the reaction of degeneration. Hyperæsthesia, but much more generally anæsthesia, sometimes only on one side, is observed, and this also may involve the special senses and affect the muscles. But muscular anæsthesia may be absent in hysteria. Rapid changes occur in the sensibility under strong electric currents, and there may be a transfer of the loss of sensation from the disordered side to the healthy side, caused by stimulating the side of the hemianæsthesia,—by mustard or by the faradic brush, or by certain metals, such as gold, or by wood,—or, indeed, by strong mental impressions.

The eye-symptoms, as Charcot has pointed out,[2] are peculiar. There may be an amaurosis, but there is no alteration of the papilla; the constricted field of vision is concentric, not, as in locomotor ataxia, star-shaped, and red is the color that is seen longest. Then in hysteria the eyebrow on the affected side is lower than on the other side, while in true paralysis it is more raised on the side affected. Nystagmus is never observed in hysteria; but hemianopsia may be met with in grave instances.

Persons affected with hysterical palsy are striking types of a nervous constitution, and, as Sir James Paget[3] mentions, show a singular readiness to be painfully fatigued by slight exertion. The palsy may seize only upon one limb, or upon part of one limb, or upon special muscles, as those of the pharynx and œsophagus, the larynx, the intestines, and the diaphragm; or it may, although it more rarely does, assume a hemiplegic or a paraplegic form. Hysterical hemiplegia presents a peculiarity in the gait, on which Todd[4] lays great stress. "In walking, when the palsy is pretty complete, the leg is drawn along as if lifeless, sweeping the ground." It is not swung round, describing the arc of a circle, as it is in ordinary hemiplegia. The palsy is almost invariably left-sided. It is apt to be conjoined to left-sided ovarian tenderness, and to very decided anæsthesia, which passes beyond the paralyzed part to the nearest portion of skin and mucous membrane, though, as a rule, still limited to the same side. Thus we find the pituitary membrane of one nostril rendered insensible, if the loss of feeling affect the face.

In hysterical paraplegia we find the same incompleteness of the

[1] Wood, Nervous Diseases and their Diagnosis, 887.

[2] International Clinics, vol. i., 2d Ser., 1892.

[3] Nervous Mimicry of Organic Diseases, in Clinical Lectures and Essays, London, 1875.

[4] Clinical Lectures on Paralysis and other Affections of the Nervous System, Lecture XIII.

palsy and the same response to electric tests already mentioned, and we are also very apt to have the symptoms of spinal irritation. Hysterical contractions of the muscles especially affect the lower extremities, though they are not uncommon in the arm. These hysterical contractures generally come on quickly, appear to be permanent, and to be associated with loss of power, but disappear as suddenly as they showed themselves. Yet they may really become permanent and combined with sclerosis of the cord, and we may find them associated with tremor, and with exaggerated knee-jerk. Ankle clonus has also been observed by Charcot as occurring in hysterical paralysis. Gowers, however, thinks that true persisting ankle clonus bespeaks secondary organic disease in the motor parts of the cord, while a spurious, irregular clonus, now ceasing, now renewed by a fresh contraction of the muscle, is characteristic of hysteria.

Very similar to hysterical paraplegia is the *paraplegia produced by hypnotic suggestion*. Charcot and Longues[1] have called attention to the fact that in this as well as in the functional paraplegia met with after railroad and other accidents, the so-called *traumatic hysteria*, there is an anæsthesia in the palsied legs which follows the fold of the groin and excludes the genital organs. The same line also enables us to distinguish between hysterical paraplegias and those of organic origin.

One of the most difficult points with reference to hysteria is to distinguish the hysterical symptoms that arise in sclerosis and in myelitis, or that follow injuries to the nervous system, from the manifestations of pure hysteria. Nothing but a careful study of the individual case, of the history, of the reflexes, of the electric reactions, of the line of the anæsthesia, of the state of the muscles themselves, laying stress on the absence of muscular wasting, also of girdle pains, and of incontinence of urine and of fæces in pure hysteria, will save from error.

Rheumatic Paralysis.—Rheumatic paralysis resembles hysterical paralysis in being ordinarily limited. It may affect any muscle or any group of muscles in the body; sometimes the rheumatic poison disorders the portio dura, and we observe, in consequence, facial palsy; or it may fasten on the radial nerve, and we have groups of muscles in the forearm palsied. Rheumatic paralysis is recognized by the history of the case; by the evidences of a rheumatic attack; by the rapid development of the palsy; by the pain which usually attends it; and by its being unaccompanied by symptoms strictly referable to a disease of the nerve-centres. It may or may not be

[1] Charcot, Œuvres Complètes, t. iii. p. 448.

attended by anæsthesia. The muscles themselves, certainly in those cases in which they, rather than a large nervous branch, are primarily and chiefly affected, are readily acted upon by electricity, unless their structure be altered; and the electro-muscular sensibility, though it may be lessened, is not abolished.

Lead Palsy.—Paralysis from lead poisoning occurs primarily, and sometimes only, in the extensor muscles of the arm, occasioning the well-known wrist-drop. It generally begins in the extensor communis, then affects the radial and ulnar extensors. Gradually other muscles become involved: there is loss of power in the ball of the thumb, in the deltoid, and in the triceps, but not in the supinator longus, or in the intercostal muscles, or in those of the lower extremities. The disturbed muscles on both sides of the body waste, entirely lose their irritability to electricity, and soon show the reaction of degeneration. The patient is weak; his movements are tremulous; he has the characteristic blue line on the gums, is obstinately constipated, is subject to colic, and lead can be found in the urine. Sometimes the poison seizes upon the brain, and epileptic convulsions and other signs of a serious cerebral affection appear, and we find marked optic neuritis. From the locality of the palsy, in addition to the accompanying symptoms and the knowledge of the man's employment, the diagnosis is usually arrived at with ease. Paralysis produced by an affection of the radial nerve shows the greatest similarity. Yet here the supinator muscles as well as the extensors, but upon one side only, are affected, which is not the case in lead paralysis, where both sides are affected and the patient can carry the hands supine. Lead palsy may be met with in children.[1]

Diphtheritic Paralysis.—Diphtheritic paralysis is a sequel of diphtheria which follows an attack of the disease within a fortnight or two months, and, therefore, after the patient is to all appearance fully convalescent. It may be very localized, merely affecting the palate or the pharynx; or very general, fastening upon both of the lower extremities, and even upon the upper. When extensive, it is ushered in by a change in the voice and a throat-palsy; there is difficulty in swallowing, fluids are regurgitated through the nose, and the saliva dribbles from the mouth. Paralysis of accommodation and strabismus and double vision are not uncommon. The paralysis of the extremities ensues gradually; day by day the muscular power is more and more enfeebled. The loss of motion is often preceded by formication, and attended by a certain amount of anæsthesia. The

[1] Cases of Sinkler, Medical News, July, 1894.

faradic electro-muscular contractility and sensibility are diminished, and the galvanic current shows mostly the same results. The knee-jerks may be abolished; and the gait may be ataxic. The palsy mends as slowly as it comes on; yet most cases fully recover. The brain itself is not directly affected; at least, there were no symptoms of cerebral mischief in the cases which have come under my observation. In some cases rupture or plugging of a cerebral vessel takes place. Commonly the palsy is due to multiple neuritis. In children affected with diphtheritic paralysis regular bulbar crises may happen.[1]

Syphilitic Paralysis.—Not unusually the syphilitic exudation is localized in the course of one or of several nerves; we have, for instance, paralysis of the sixth or paralysis of the fifth with or without paralysis of some other cerebral nerve. But as syphilis attacking the nervous system is chiefly characterized by a want of uniformity in the lesions it produces, so we observe dissimilar phenomena, preceding or attending the palsies. Thus, we do or do not, though in point of fact we usually do, find the paralysis associated with pain in the head, with optic neuritis, with sleeplessness, vertigo, impaired memory, and sickness at the stomach. Decided vertigo is prone to take place where the syphilitic affection has led to disease of the vessels, and is apt to be the forerunner of local softening and of hemiplegia. When disease of the membranes has happened, headache is severe, and local spasms or convulsions occur. The same symptoms are encountered when there is a growth in the hemisphere, which is very apt to be near the surface; though here again the form of mischief may be comparatively latent, the patient may have only occasionally convulsions, and the paralysis be slight or improving, yet a fatal coma may follow a few convulsions. Instances of this have come under my observation.

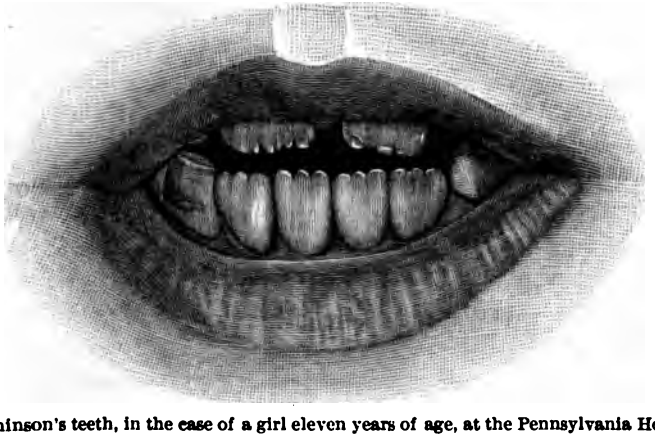
But, as a rule, syphilitic paralysis does not terminate fatally. In truth, the ease with which the palsy and its attending phenomena mostly yield to treatment, forms one of the traits of the malady. Other common features are—that it ordinarily affects persons younger than those in whom we find paralysis dependent upon disease of the nervous centres, and especially of the brain; and that its manifestations are shifting and capricious, and rarely symmetrical. These same signs characterize syphilitic affections of the nervous system in which paralysis is not among the symptoms. Paralysis of the third nerve is a frequent result of syphilis; but, as already stated, the poison

[illegible]
[illegible]

may attack any part of the nervous system, and paraplegia dependent upon disease of the cord is not very uncommon. A progressive multiple palsy of cerebral origin, clearly affecting dissociated muscles, is usually syphilitic, and is mostly due to several patches of gummatous meningitis. At times a rapid, almost universal paralysis, as Buzzard notices, occurs in syphilitic subjects. This is most likely of peripheral origin. It is among the peculiarities of syphilitic palsy that the lost electro-muscular contractility returns rapidly.[1]

Erb has called attention to an association of symptoms regarded as characteristic *syphilis of the cord.* They come on gradually at first, but may then rapidly develop. They are increased reflexes, with but little muscular rigidity, slight spastic gait and muscular weakness, disorder of the bladder functions, and disturbances of sensation in the legs in the form of paræsthesia. The symptoms are confined to below the waist.

FIG. 14.



Hutchinson's teeth, in the case of a girl eleven years of age, at the Pennsylvania Hospital.

In syphilis the mischief to the nervous system may not happen for years after the infection, of which the history is often very obscure. The disorder may be the result of an *inherited taint.* But such cases cannot be recognized unless there are other signs of syphilis than the suspected nervous symptoms; and chief among these signs are the evidences of periostitis in the long bones and of disseminated choroiditis in the fundus of the eye. Then there is that valuable test of congenital syphilis discovered by Mr. Hutchinson,—a malformation of the two upper central permanent incisors, which consists in their being narrower at their cutting edges than at their insertions, and often notched. The same observer has called attention to diffused

[1] Engle, Philadelphia Medical Times, Dec. 1877.

opacity of the cornea and to diseased nails as being common among the manifestations of the inherited disease. Paralysis also may occur, as in the case reported by Bartlett;[1] but it is very rare.

LOCAL PALSIES.

The forms of paralysis which have just been noticed are mainly such as are designated as partial. When the loss of power is very limited, the palsy is spoken of as *local;* most of these local palsies are peripheral, and the result of neuritis.

Facial Palsy.—Of the local paralyses, of particular importance from its frequency, is facial, or Bell's palsy. The disease consists in an affection of the portio dura of the seventh nerve. In consequence of the derangement of this motor nerve, nearly all the muscles of one side of the face lose their faculty of motion, and, as it is their play which gives expression to the countenance, the appearance of the face is extraordinary. The eyelids are open and fixed; the features are rigidly composed on one side of the face, but reflect every change of feeling on the other: the mouth is distorted, being drawn to the unaffected side: the naso-labial fold is effaced: the eye waters; and in the old the furrows disappear from the forehead. In some cases the velum palati is involved in the paralysis. The impaired muscles waste: their electric irritability is diminished and degenerative reactions may be present. Sensation remains unaltered so long as the fifth nerve is not disturbed.

The causes of the palsy are such as influence the distressed nerve in its course or at its periphery: a wound: mumps: ear-disease: exposure to cold: rheumatism: syphilis. The most common cause is a neuritis from cold affecting the nerve within the Fallopian canal. The malady is easily discriminated from the facial palsy of disease of the brain by the inability to close the eyelids, owing to the paralysis of the orbicularis palpebrarum: by the absence of headache, of vertigo, of mental confusion, of loss of memory: by the much more complete though strictly local character of the paralysis, the affected muscles even failing to participate in bilateral or emotional movements: and, except in slight lesions of the nerve, by the lost electro-muscular contractility. In severe cases, indeed, the muscles soon cease to respond to faradization, while the galvanic irritability is preserved and even heightened, and the reaction of degeneration is very marked. Electric stimulation of the diseased nerve shows that it quickly loses its excitability, both to faradism and to galvanism.

[1] Clinical Society's Transactions, vol. xi.

The observations of Erb enable us to tell with considerable accuracy the exact part of the nerve affected. They take into account well-known anatomical and physiological facts, and lead to these conclusions. If there be complete palsy of all the facial branches with the exception of the posterior auricular nerve, the lesion is in the main trunk of the facial, exterior to the Fallopian canal. If the auricular nerve be also implicated, the lesion is within the Fallopian canal below the origin of the chorda tympani, the most common seat of the affection. If taste and salivary secretion be disturbed on the side of the tongue corresponding to the palsy of the face-muscles, the lesion is between the points where the chorda tympani and the tympanic branch are given off. If in addition the sense of hearing be abnormally increased, we may infer that the nerve is affected between the tympanic branch and the geniculate ganglion, and at the latter point palsy of the palate is superadded; and higher, up to the entrance into the brain, disorders of taste happen. Eventually implication of other cranial nerves, as of the auditory, also occurs.

Cases of facial-nerve palsy generally recover. Sometimes, however, the recovery is incomplete, and a rigidity with some contraction of the affected muscles takes place, which, when slight, may make the sound side appear relaxed, and the diseased side seem the normal one.

In rare instances the facial palsy is on both sides. Now, in this *double facial palsy* the lesion may be within the cranium, such as compression by a tumor, or may affect the nerves while passing through the medulla and pons in their farther course. When dependent simply on a local affection, and therefore limited to the manifestations of paralysis of the portio dura, we find the same causes at work which give rise to the one-sided disease. Exposure to cold and rheumatism are the most frequent; but syphilis is also among them. In an instance detailed by Todd, in which there was disease of the temporal bone, the portio mollis was also implicated. The face is immovable, or nearly so, and the palsy is generally more complete on the left side than on the right. The muscles do not respond to electricity, or respond imperfectly, and we notice, as in the one-sided malady, that a continuous current may excite their action, while faradization does not. Nay, the two sides may give different results in this respect,[1] most likely caused by different conditions of exudation and of pressure on the affected nerves.

[1] Case of Baerwinkel, Schmidt's Jahrbuch, Bd. cxxxvi. No. 1.

Paralysis of the Nerves of the Arm.—Paralysis of one or more nerves of the arm is very often encountered. It may happen from rheumatism, from cold developing a neuritis, from traumatism or fracture, or from the pressure of a growth; but its most common cause is accidental compression. A person falls asleep with his head on his arm, and a temporary palsy results; or it may follow the use of a crutch. In truth, the disorder may be taken as the type of the *palsies by compression*, and we find that the electro-muscular contractility depends on the severity of the nerve-lesion; as a rule, there is reaction of degeneration. Sensory symptoms are slight or wanting; often there is numbness or tingling.

The nerve most frequently paralyzed is the *musculo-spiral*, or its main branch the radial, and we observe palsy of the extensors of the wrist and the fingers, and of the supinators. In the loss of power in these muscles, in the mode of onset, and in the unilateral affection we find the differences between the palsy under consideration and the wrist-drop of lead palsy. When the *median* nerve suffers, the pronators, the radial flexor of the wrist, the flexors of the fingers,—except the ulnar half of the deep flexor,—the abductor and flexors of the thumb, and the first and second lumbricales are paralyzed; while sensibility is impaired or lost on the palmar aspect of the thumb, the index and middle fingers, and adjacent portions of the ring-finger, and often on the dorsal aspect of the last phalanx of the index and middle fingers.

Involvement of the *ulnar* nerve shows itself in palsy of the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the interossei, the third and fourth lumbricales, the adductor and inner head of the short flexor of the thumb; and impairment of sensibility in the parts of the hand and fingers not supplied by the median and radial nerves. From those diseases of the spinal cord which begin with arm palsy, the local malady is distinguished by the tenderness in the course of the nerve, and the one-sided paralysis. The same signs separate this arm palsy from the loss of power in the wrists, arising from atrophy of the muscles in the overworked parts, occurring in undernourished persons, as in poorly fed and hard-worked shoemakers.[1]

About other local palsies, as of the pharynx and œsophagus, of the larynx, of one side of the palate, of the tongue, of the muscles of the eye, of the diaphragm, of isolated muscles of the trunk, and of the extremities, it is impossible here to enter into particulars. But there

[1] Chambers on the Indigestions.

are some forms of local palsy which, from their striking interest, it is necessary to describe, the most important of which is the paralysis of the tongue and parts concerned in deglutition.

Bulbar Paralysis.—Bulbar palsy can scarcely be considered a local palsy. It has a close relation to progressive muscular atrophy, yet, from a clinical point of view, its main manifestations are those of disorder of special nerves. In this bulbar or *glosso-labio-laryngeal* paralysis, the first symptoms which are likely to attract attention are, that the tongue seems less supple and the utterance becomes nasal or thick, the food lodges between the teeth and cheek, and the saliva dribbles from the lips and corners of the mouth. As the paralysis progresses, articulate speech is almost lost, as is the reflex action in the throat; the shape of the tongue is altered, it generally dwindles, and at times shows twitching of its fibres, or lies motionless in the mouth; the posterior nares can no longer be closed by the velum and muscles of the posterior palatine arch; deglutition becomes very difficult, and the patient is tormented with hunger. Reflex irritability of the mucous membrane of the larynx is frequently lost; the respiratory movements are unusually weak, and fits of suffocation ensue. The general debility becomes extreme, and the patient is apt to perish by the sudden stoppage of the heart's action. The disease is unmistakable. Double facial palsy resembles it most; but here the tongue is not involved, and the eyelids remain open; on the other hand, in bulbar paralysis the lower part of the face only is motionless.

This condition must be distinguished from so-called *pseudo-bulbar palsy*, depending upon bilateral inflammatory or destructive lesions of the cortical centres for lips, tongue, and pharynx, or their centrifugal paths. The acuteness of onset, perhaps with apoplectic phenomena, the absence of wasting and of electrical alterations, and the presence of other symptoms, such as hemiplegia of ordinary or of alternate type, differentiate the cerebral from the bulbar affection. Symptoms of bulbar palsy may, however, result from an acute lesion, such as hemorrhage, inflammation, or softening of medullary nuclei, and be sudden in onset. The chronic affection is generally of rather slow development and slow but relentless progress; but it is not nearly so chronic a malady as progressive muscular atrophy, which may last from ten to twenty years, while the bulbar paralysis has, like lateral sclerosis, an average duration of from one to three years.[1] Progressive bulbar paralysis has its seat of lesion in the medulla oblongata, in the motor nuclei, which undergo a degenerative atrophy;

[1] Möbius, Schmidt's Jahrbuch, No. 2, 1882.

and we understand the main symptoms when we reflect on the nuclei which connect the hypoglossal, the spinal accessory, the vagus, and the facial.

Under the designations *myasthenia gravis pseudo-paralytica* and *asthenic bulbar paralysis* a condition has been described characterized by weakness of voluntary muscles, especially of those controlled by the bulbar nerves, or by undue fatigue after ordinary activity, without wasting or without changes in reflexes or in sensibility. Remissions and exacerbations are common, and may occur suddenly. The affected muscles respond normally to electric stimulation except that to tetanizing currents the response grows gradually feebler and feebler. The affection is believed to be of toxic origin.

With reference to all these local palsies we are sometimes much perplexed to know if the palsy be the result of beginning disease of the brain or spinal cord, or if it be purely local. To speak first of the brain: the cerebral symptoms may not be marked, or they may be so contradictory as to afford no real help in diagnosis. When, however, we discover, as we generally can, that the palsy affects muscles which are supplied by different nerves and such as have no communication with one another, we may set down the complaint as having a central origin. As regards the distinction from spinal affections, the almost constantly single-sided character of the symptoms in local palsies, and their double-sided character in spinal affections, are very important. The strikingly symmetrical kind of the palsy and the element of pain are features of great diagnostic significance in the wide-spread peripheral paralyses as seen in multiple neuritis.

PALSIES CONNECTED WITH MARKED MUSCULAR WASTING.

There is a group of palsies especially marked by wasting of the muscles. In some affections already discussed we have found wasting among the symptoms, as at times in myelitis, and in cervical pachymeningitis with considerable damage to the nerve-roots, where atrophy of the arms happens. Again, atrophy of the muscles of the trunk and limbs is often met with in the advanced stages of progressive bulbar paralysis. But in all these affections there are more distinctive symptoms. In some affections the wasting of the muscles is the pre-eminent feature. This is particularly the case in progressive muscular atrophy and in the essential paralysis of childhood.

Progressive Muscular Atrophy.—This form of "wasting palsy" is due to chronic or subacute degenerative changes in the gray matter of the anterior horns of the spinal cord, particularly the

large ganglion-cells, sometimes in association with similar changes in the peripheral motor nerves and the pyramidal tracts. The affected muscles undergo atrophy of varying degree and extent.

Progressive muscular atrophy is a disease of adults, and essentially of men who use their muscles continuously and violently. Its most striking sign is increasing inability to perform certain movements. When the muscle chiefly concerned in the attempted motion is examined, it is found to have dwindled. Soon other muscles follow; and their wasting, too, is accompanied by further muscular weakness. The disorganizing muscles twitch, and tapping them sharply causes a marked contraction of the fibres. These muscles of the face, as a rule, escape. In the affected part the circulation becomes languid; it is also very susceptible to cold, and its temperature is lowered; there is a feeling of numbness in it, but rarely pain; to pressure it is soft and yielding. The muscles most frequently attacked are those of the hand, the flexors and supinators of the forearm, the biceps, the deltoid, and the other muscles of the shoulder. Sometimes the disease begins in the trunk and the lower extremities; but it is most common to have it marked in the upper extremities and to find only weakness and spasm in the lower. Sometimes, also, bulbar symptoms, with weakness of the muscles of the lips and tongue and of the pharynx and larynx, appear, and changes are found in the medulla analogous to those present in the cord. The decrease of the muscular fibres gives rise to strange and palpable deformities, and, when the muscles of the trunk are involved, to extraordinary positions of the body, in consequence of all antagonism to the healthy muscles having been removed.

In the parts affected the reflex action is lost; even the deep reflexes disappear. We see this happening with the knee-jerk just so soon as the muscles of the legs become flaccid and begin to waste. To the electric currents, both faradic and galvanic, the muscles respond feebly; still they respond, and in portions where there are many sound fibres they contract energetically. The degree of response depends, indeed, on the degree of disorganization and wasting. Excitability to the galvanic current remains much longer than that to faradization; the reaction of degeneration is likely to be present.

From *cerebral hemiplegia* progressive muscular atrophy differs by its much more gradual invasion, by the rapidity but want of uniformity of the muscular atrophy, by the lost reflexes, by the diminished electric excitability, and by the absence of disordered intellect and of other signs of disease of the brain. Difficulty in articulation and in deglutition may occur in either. From *general spinal paralysis* it is

diagnosticated by the spinal malady affecting primarily all the muscles of the lower extremities before those of the upper become involved. Then, too, if the spinal paralysis be due, as it so generally is when extensive, to myelitis, the alterations of sensibility, the totally lost electro-muscular contractility, and the affection of the sphincters are striking traits of difference.

The difficulty of distinguishing cases of *local paralysis* from progressive muscular atrophy is at times very great. Yet generally we may separate the latter, for instance from rheumatic palsy, by noticing that this affects a group of muscles rather than one muscle, or than one muscle here and another there. Further, the atrophied muscle in the rheumatic disorder is the seat of pain intensified by movement, and it contracts well under the electric stimulus. The same test by the electric current is of service in discriminating the muscular disease from hysterical paralysis, and from paralysis consequent upon injuries of nerve-trunks and upon lead poisoning. In the first of these palsies the electrical contractility is, except temporarily in cases of old standing, intact, in the others it is abolished; in progressive muscular atrophy, save when the wasting is extreme, it is simply enfeebled. Besides this, we attach importance to the unimpaired sensibility, the capricious and unequal manner in which the atrophy seizes upon the muscles in this malady, the fibrillation, and the beginning of the wasting in the thenar muscles and the interossei.

Hirst[1] points out the occurrence of muscular atrophy as a phenomenon of *hysteria*. The peculiarity of this form of wasting is its unilateral or circumscribed character, though sometimes it is general. The recognition depends upon the psychic state of the patient and the occurrence of hysterical or hystero-epileptic convulsions.

The muscular atrophy due to degeneration of the anterior horns of the spinal cord differs from that due to *multiple neuritis* in its progressive rather than retrogressive character, but especially in the absence of symptoms of sensory derangement.

The most difficult differential diagnosis we may be called upon to make is to distinguish certain cases of progressive muscular atrophy from *bulbar paralysis*. In truth, the two affections often coexist. The diagnosis depends upon the distribution of the symptoms, the morbid process being essentially the same in the two sets of cases. In the one the arms, and sometimes also the legs, suffer: in the other the tongue, the lips, the pharynx, and the larynx. Defective

[1] Deutsche Medicinische Wochenschrift, 1894, No. 21, p. 459.

pronunciation points to the bulbar malady. Failure of the respiratory power is common to both.

Local atrophies may be mistaken for part of the general disease. There is, for instance, an affection, *unilateral progressive atrophy of the face*, in which gradual wasting of one side of the face occurs, of the soft parts first, and then of the deeper tissues. The facial hemiatrophy follows blows and contusions, abscess of the ear, influenza, typhoid fever, or, as in Cohen's case, an attack of erysipelas. It begins with a discoloration of circumscribed spots, a white or yellowish discoloration; the subcutaneous fat disappears, and the beard and eyelashes change. Sensation is, as a rule, not affected, nor are the electrical reactions changed.[1] But in progresssive muscular atrophy the face almost always escapes; if it be affected, it is so on both sides. *Acute or chronic joint-inflammations* are attended with weakness and wasting of the muscles moving the affected parts. The extensors usually suffer, occasionally also the flexors, and rarely distant muscles. Another limited atrophy is a wasting from *overuse of muscles*, seen especially in the small muscles of the hand. It shows no tendency to extend.

Paralyzed muscles atrophy, and may subsequently undergo degenerative change; but the distribution differs from that of progressive muscular atrophy, and we lay stress on the symptoms that usher in and that attend the paralytic state.

In the condition known as *syringomyelia*, in which the central gray matter of the spinal cord is replaced by gliomatous tissue that breaks down and gives rise to the formation of a cavity, we have fibrillar contractions in the affected muscles and atrophy, with resulting deformities. But symptoms of sensory derangement appear earlier and are more pronounced. Common sensibility is generally unchanged, where there is inability to distinguish heat and cold, and often also to appreciate pain. The sphincters are not disturbed; the knee-jerks are normal or exaggerated. The muscles waste, rapidly lose their faradic excitability, and the reaction of degeneration is finally established. The symptoms, on the whole, are of slow development, and show themselves chiefly in the arms and in the upper part of the trunk. There is unsteadiness of motion, with muscular weakness rather than paralysis, and trophic disturbances in the skin, such as thickenings, eruptions, ulcerations, are marked; so are arthropathies. In the legs

[1] See cases, Journal of Nervous and mental Diseases, New York, March, 1880; Schmidt's Jahrbuch, No. 7, 1881; St. Louis Alienist, April, 1881; and Skyrme, Brit. Med. Journ., March, 1892.

there may be spastic paresis. Inequality of the pupils and nystagmus are not unusual. Deviation of the spine is common; it was present in half the cases analyzed by Bruhl.[1] A large number of cases originate in injuries to the back.[2] A case presenting symptoms of syringomyelia has been recorded in which after death gummata were found on either side of the brachial enlargement of the cord.[3]

The disorder described by Morvan and called by his name, and also "painless whitlows," presents symptoms of syringomyelia in conjunction with those of peripheral neuritis. At first there may be neuralgic pains in the hands, followed by anæsthesia and muscular wasting, and the formation of whitlows that undergo ulceration, and are attended with necrosis of the phalanges. The altered vasomotor condition is also shown by the elevation of temperature in the weakened limbs, the red spots or the intense flushing of the surface, and the ease with which the skin blisters.

There is another disease resembling progressive muscular atrophy which may be here mentioned, the singular affection endemic in parts of Japan, known there as *kakke*, and probably identical with the disease called in India and Brazil *beriberi*. The generally accepted view is that beriberi is an infectious disease, developing under conditions of high temperature and moisture, and presenting the symptoms of a multiple neuritis. Observations made in Japan render it likely that the cause of the neuritis is generally poisoning by damaged rice, and it is said that attention to the diet has almost banished the disease from the Japanese navy.[4] It has also been thought to be due to absence of fat from the dietary. Four types of the malady are recognized,—an incompletely developed or rudimentary form; an atrophic form; a dropsical form, with or without atrophy; a pernicious or cardiac form. The most conspicuous symptoms are impairment of motion, with wasting and diminution in mechanical and electrical irritability, sensory changes, circulatory disturbances, abolition of the knee-jerks, diminished secretion of urine, and albuminuria.

A form of progressive muscular atrophy, known as the *peroneal type*, and described by Charcot, Marie, and Tooth, usually sets in early in life, affecting first the muscles of the foot and leg, sometimes those of the hand and forearm, and extending upward. In addition to weakness and wasting, sensation is deranged and degenerative elec-

[1] Étude de la syringomyélie, Paris, 1890.

[2] Guy Hinsdale, Syringomyelia, Philadelphia, 1897.

[3] Beevor, Lancet, vol. ii., 1893, p. 1252.

[4] Takaki, Report of the Japanese Navy, 1886, quoted in Sajous's Annual, vol. i., 1888.

tric reactions are present. The condition is dependent upon neuritis and it occurs in families. Club-foot is a common resulting deformity.

It is sometimes a matter of extreme difficulty to distinguish cases of what are called *progressive muscular dystrophy*, where there is no appreciable central nervous lesion, from the progressive muscular atrophy under consideration. When the former disease happens in children the distinction is not so difficult; for the age, and the circumstance that not infrequently several members of the family are affected, in some of whom it may assume the pseudo-hypertrophic form, show what it is. But in adults there may be great uncertainty. The extremely slow progress of the disease; its not unusual beginning in childhood; the fact that the muscles of the forearm and hand escape, as a rule, while the face is sometimes involved, as well as the latissimus and the lower half of the pectoralis, that it affects males far more commonly than females, and that it is congenital, are some of the characteristic points. Fibrillary twitching of the muscles is wanting, the deep reflexes are enfeebled, and the electric reactions undergo only quantitative diminution proportionate to the degree of wasting.

Several types of the disease have been described, the *idiopathic*, the *pseudo-hypertrophic*, the *juvenile* or *scapulo-humeral*, the *infantile* or *facio-scapulo-humeral*, and the *hereditary*, but the distinctions are not readily maintained. All present in common hereditary or family distribution, onset early in life,[1] preponderance among males, progressiveness of course, weakness and wasting, sometimes preceded by apparent hypertrophy of various muscles, lessening of mechanical and electric irritability and of deep reflexes. The gait is peculiarly waddling, and extraordinary attitudes are assumed in attempting to rise from the ground. The lesions in the muscles consist in increase in size of some fibres, with diminution of others, degenerative changes, and more or less increase in the interstitial connective and fatty tissues. The wasted muscles undergo shortening and contraction, and various deformities result. The function of the sphincters is, as a rule, preserved; intelligence is not affected; and sensibility is unimpaired.

Infantile Paralysis.—In this disease, also known as essential paralysis of children, and acute anterior poliomyelitis, rapid wasting of the muscles is the striking feature. It is pre-eminently an affection of early childhood, and, as shown by Wharton-Sinkler, occurs much

[1] Destarac, La Médecine Moderne, 1894, No. 89, p. 1387, has reported a case of pseudo-hypertrophic paralysis in a man sixty-eight years old, without hereditary predisposition.

more commonly in summer than in winter. It happens most frequently during the first dentition, and is often ushered in by fever, by diarrhœa, nausea or vomiting, and by convulsions. The palsy comes on quickly, generally before the fever-disturbance has passed away; or an entire limb, or even both legs and arms, may almost from the onset be affected. In any case the palsy becomes plainly discernible as the fever subsides. It is apt to begin in one limb and in a few days to become wide-spread. But it disappears, except from a particular region in which the muscles quickly waste.

Yet the palsy may at first shift; it passes away from some limbs, or fixes upon others or upon different groups on different sides of the body. It rarely, however, remains as palsy of more than one side, and is not associated with loss of sensibility. There is often decided recovery within six months from the onset of infantile paralysis; although some loss of power may be permanent. The affected muscles are apt to begin to atrophy after the paralysis has lasted a month, and when their wasting is marked they no longer respond to the faradic current, though they may still react strongly under the galvanic current; but gradually this excitability, too, is lost. Both the superficial and tendon reflexes are lowered or abolished. After six months or a year some faradic irritability is apt to return. The functions of the bladder and rectum are very seldom affected. In protracted cases, permanent shortening of muscles happens, contraction of the joints takes place, and atrophy of portions of the osseous system occurs, or rather a want of its development in the blighted parts, and various and striking deformities result.

Now, the onset of these cases, the febrile symptoms, the occasional retrocession from certain parts, and the subsequent course, separate infantile paralysis from progressive muscular atrophy. Then in forming a diagnosis we may take into account the extreme rarity with which children are attacked with progressive muscular atrophy. Yet the affection may happen in children, and then as Duchenne pointed out, is apt to show itself first in the muscles around the mouth. On the other hand, we must not forget that a disease identical with the essential palsy of children is met with in adults. Beginning acutely with febrile symptoms, headache, vomiting and disordered [illegible] it passes with [illegible] of muscles [illegible] without impaired sensibility. [illegible] passing [illegible] shows some [illegible] and passing [illegible] without [illegible] and has the [illegible]

of the cells of the anterior horns. With reference to this acute atrophic spinal paralysis or *acute anterior poliomyelitis*, we have learned that often complete or nearly complete recovery from the threatening symptoms takes place, and that it is probably due to a systemic infection.

From the foregoing remarks it might be inferred that children are only subject to palsies that are spinal. But this is not the case. We find in them a whole group of *cerebral palsies*,—not nearly so frequent, it is true, as the spinal group, but palsies in which the lesion is cerebral, extending from any part of the cortex to the pyramidal tracts of the cord, and broadly distinguished from the spinal palsy by heightened reflexes, unchanged electrical reactions, loss of power with disordered movements or spasm, and retarded growth of the affected parts. We may find either hemiplegia, bilateral hemiplegia, or paraplegia as the form of paralysis. In some instances the affection follows delivery with the forceps; like spinal infantile palsy, it has been observed after infectious diseases. Under the first condition it is probably due to meningeal hemorrhage; under the second, to either hemorrhage into brain or membranes, or to vascular occlusion. Sometimes the disease begins with fever accompanied by convulsions; these may be followed by marked coma. The hemiplegia is most persistent in the arm, and is apt to be associated with spastic contraction, producing a peculiar gait. Post-hemiplegic chorea and mobile spasm and athetosis were observed in a considerable number of cases analyzed in Osler's elaborate monograph.[1] Convulsive seizures on the paralyzed side or general epilepsy are yet more common, and the intelligence is enfeebled.

In the bilateral form of hemiplegia the legs are more involved than the arms; spastic contractions of the muscles of the extremities are most marked; the mind is very much affected; sensation is not disordered. Destruction of the motor centres of the cortex is the essential lesion in bilateral spastic hemiplegia.[2] In the spastic cerebral paraplegia of children McNutt[3] found descending degeneration in the pyramidal tracts; the disease is limited to the lower extremities; there is no muscular wasting; the gait is stiff or cross-legged. The malady usually exists from birth, and follows a difficult labor. The

[1] The Cerebral Palsies of Children, 1889. See, also, Sachs and Peterson, Study of Cerebral Palsies of Early Life, based upon one hundred and forty cases. Journal of Nervous and Mental Diseases, May, 1890; and Sachs, Samml. Klin. Vortr., No. 46, 1892.

[2] Osler, *op. cit.*

[3] Amer. Journ. Med. Sci., vol. i., 1885.

intellect is impaired, though not always markedly so. Wood[1] states the affection to be the result of sclerotic and atrophic changes in the brain.

Before proceeding, we will examine the main forms of paralysis which we have been studying, arranged in a tabular form, and chiefly with the view of ascertaining the seat of lesion, premising that the statements must be received rather as generally true than as absolutely so.

TABULAR VIEW OF PARALYSIS.

Symptoms.	*Seat of Lesion.*
Inability to move leg and arm of one side. Sensation unimpaired, unless posterior third of posterior limb of capsule involved. Paralysis of muscles of lower part of face; mouth drawn towards healthy side. Electro-muscular contractility preserved. Reflex excitability of the tendons exaggerated.	Corpus striatum, involving internal capsule, both on side opposite to the palsy.
Same symptoms, dependent on involvement of internal capsule. Mobile spasm and incoördination in paralyzed parts.	Optic thalamus.
Same symptoms, but paralysis of face, with anæsthesia, on opposite side to that of arm and leg, and usually marked; conjugate paralysis or spasm of eyes; difficulty in deglutition and articulation. Heightened temperature; convulsions; contracted pupil. Urine may contain sugar or albumin. Early rigidity of paralyzed muscles.	Pons Varolii, on side opposite to palsy of limbs. The part affected is below decussation of facial nerve.
Same symptoms, but face paralyzed on both sides.	Pons Varolii, and at level of decussation of facial nerve.
Paralysis of arm and leg and lower part of face on one side; third nerve paralyzed on other side; defective sensation; vasomotor disturbance.	Crus cerebri on side corresponding to paralysis of third nerve.

[1] Nervous Diseases and their Diagnosis.

TABULAR VIEW OF PARALYSIS.—*Continued.*

Symptoms.	*Seat of Lesion.*
Paralysis of motion of face, arm, or leg, soon followed by rigidity; sensation may be impaired. Reflexes, superficial and deep, increased. Convulsions.	Cortical part of brain in motor zone on side opposite to palsy.
Motion more or less completely affected on both sides of body, except in face; paralysis of hypoglossal, glossopharyngeal, and spinal accessory nerves; often rapidly fatal.	Medulla oblongata.
Both legs and lower part of trunk paralyzed as to motion; loss of sensation; some wasting of muscles; loss of power over bladder and rectum; reflex excitability in legs heightened, trunk reflexes impaired; electric contractility diminished or lost; trophic changes; paralysis of muscles of respiration in some instances.	In the cord throughout its section above the lumbar enlargement, as in transverse myelitis of the dorsal cord.
Both legs paralyzed, muscles of legs flaccid; feet extended; anæsthesia; incontinence of urine from the start. Superficial and deep reflexes lost. Rapid wasting of muscles. Reaction of degeneration. Trophic changes.	In the cord in lumbar enlargement, as seen in myelitis of these parts.
Arms as well as legs paralyzed; arms flaccid, legs spastic; otherwise symptoms much the same; affection of pupils.	Cervical region of the cord, as in cervical myelitis.
Paralysis irregular in degree and distribution, relaxation of muscles, sensation unimpaired, only transient loss of control over bladder and rectum; marked lowering or extinction of reflex excitability in the palsied muscles and tendons; lost electro-muscular contractility to faradic current; usually reaction of degeneration; rapid muscular atrophy; no bedsores; if disease become chronic, muscular contractions.	Anterior horns of the cord, as in degeneration of the cells in acute poliomyelitis.

Ataxia.

Loss of co-ordination of muscular movement, which in the legs shows itself especially in the gait, and in the hands in the difficulty of executing delicate movements, but which strangely contrasts with the muscular power that is present, is found in general paralysis of the insane, multiple neuritis, and diphtheritic paralysis. But the ataxia is most constant and marked in locomotor ataxia.

Locomotor Ataxia.—In this disorder we have uncertainty of motion and seeming palsy; or, in the words of Duchenne, who gave it the name of progressive locomotor ataxia, it consists in "a progressive abolition of the co-ordination of movement with apparent paralysis contrasting with the integrity of muscular force." The patient is not deprived of the power of motion, but of the power of controlling his motion: hence he staggers in his walk, or cannot walk at all without support; the muscles are obedient to the will, but the peripheral impressions by which motor impulses are guided are improperly or imperfectly conveyed.

Locomotor ataxia is identical with a form of palsy clearly recognized by Todd, and with the malady described by Romberg as *tabes dorsalis;* from the lesion it exhibits, it is often called *posterior sclerosis*, degeneration of the posterior columns of the cord, and of the posterior nerve-roots being its main cause. A wasting of the nerve-fibres of the peripheral spinal sensory nerves has also been found.

The affection is a very chronic one, lasting many years. It is a disease of adult life, and it occurs far more commonly in men than in women. It may originate without assignable cause, or may follow alcoholic excess, or exposure to cold, or injury or inflammation of the cord, or is hereditary. It has been observed to follow pernicious anæmia.[1] It is most frequently found to be associated with a history of syphilis. Among its early symptoms are piercing pains, lightning-like or similar to electric discharges, in the lower extremities; enfeeblement or loss of knee-jerk; disordered gait; diplopia or other disturbances of vision, which may be attended with the "Argyll-Robertson pupil,"—a small pupil that does not respond to light, but does respond to accommodation,—or with paralysis of the sixth or the third pair; and a zone in which sensation is greatly impaired on a level with the third, fourth, fifth, or sixth dorsal vertebra.[2]

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[1] Putnam, Amer. Journ. Med. Sci., March, 1895; also Burr, University Medical Magazine, April, 1895.

[2] Hitzig, in Ziemssen's Cyclopædia, article "Atrophy of Brain."

Following these phenomena, or making its appearance with them, is a difficulty in co-ordinating movements and in maintaining the equilibrium of the body. It is manifest in attempting to walk with the eyes closed or in the dark; and the patient is unable to take a step, or to stand erect with his feet in juxtaposition, without swaying and losing his balance. This, the so-called *Romberg* symptom, is not pathognomonic, but it is very valuable in the diagnosis of the earlier stages, and so is the difficulty in placing the foot on small surfaces, in buttoning the clothes, or in walking backward. Nor can the patient stand upon his toes, or upon one foot. Another symptom is *Fraenkel's* symptom, or hypotonia, the power to straighten the legs completely when at right angles to the body.

Yet the stumbling gait is not connected with true paralysis. The muscles can act vigorously, are well nourished, contract readily when faradized, except in advanced stages of the disease, and show neither tremor nor spasm. The feet, in walking, are raised high in air and brought down upon the heel or upon the whole sole. The cutaneous reflexes are generally, yet not always, impaired; there is absence of the patellar tendon reflex in both knees. Sensibility is markedly diminished, pinching and pricking the foot may scarcely be felt, contact with the floor may not be appreciated, perception of sensory impressions may be delayed, girdle-sense is often present, and the tactile sensibility may be almost gone; but all kinds of curious sensations are complained of. The power to appreciate differences of temperature may, though it does not always, remain, and there is a delay in the perception of pain. The muscles, too, lose their sensibility. It is not unusual to have pains in the region of the fifth nerve. The intellect is unimpaired, unless frequent attacks of vertigo and epileptic seizures should be among the symptoms. The eyesight fails more and more, there is loss of color-vision, and an atrophy of the optic nerve may produce irremediable loss of sight; the hearing, too, may become much affected; and signs of valvular disease of the heart, especially of the aortic valve, show themselves. The functions of the rectum and bladder are not markedly disordered, though retention of urine and sluggish action of the bladder are not infrequent. The sphincter ani is often weak, but constipation is common. There is loss of sexual power. Dropsy and local sweating are met with, and so is swelling of the joints, without redness and usually without pain.

But the joint affection may appear, as Charcot has taught us, before the loss of power of co-ordinating movement. In time, it may be rapidly, the articular extremities of the bones disappear, and the

joints undergo a kind of dislocation. The shafts of the bones, too, show defects of nutrition, and spontaneous fractures happen. The teeth drop out of the atrophied alveolar processes, and so may parts of the bones themselves;[1] the tendons tear; the tongue may dwindle on one side; the spine becomes curved. Herpetic, bullous, and pemphigoid eruptions or ecchymoses may appear during or subsequent to exacerbations of the lightning pains. Perforating ulcer of the foot has also been observed among the trophic changes.

Among some of the less common symptoms is drooping of the eyelids, accompanied by weakness of all the muscles attached to the eyeball, and a sense of the face being covered by a mask.[2] Another symptom, more frequent, is the occurrence of spasms and pain in the epigastric region, with attacks of vomiting. These gastric crises, as they have been termed, may be found to happen in those who complain much of fulness in the abdomen and of unsatisfied hunger. They have even been known to lead to vomiting of blood. Buzzard[3] shows the symptoms to be dependent upon sclerosis affecting the nucleus of the vagus. There is always in these gastric crises acid fermentation, but hydrochloric acid is also constantly found.[4] There are at times attacks of laryngeal spasm in ataxics. Arthropathies often happen in those who present laryngeal or gastric crises.

These two forms of crises are by far the most frequent. But, in addition, we have intestinal crises, urethral crises, rectal crises, genital crises, renal crises, cardiac crises, and others, in which, as the chief symptom, violent paroxysms of pain occur, that pass away and are found not to be connected with any organic change of the seemingly diseased part. The true meaning of these pain crises, as well as the distinction from the visceral affections they simulate, is detected in the absent knee-jerk and in the other symptoms of the ataxic malady.

In considering the diagnosis of locomotor ataxia, let us first examine how it differs from *general paralysis* of the insane. Both maladies are very chronic in their course, and in both there is loss, or certainly impairment, of muscular co-ordination. In the one case, however, it exists with tremors, with thickness of speech, with dementia, with peculiar delusions, with exaggerated knee-jerks. Then, in locomotor ataxia, the hands are rarely affected; indeed, should, in process of time, the upper extremities share in the disorder, there is

[1] Newmark's case, Medical News, Jan. 26, 1895.

[2] Hutchinson, Transact. Royal Medico-Chirurg. Soc., 1879.

[3] Diseases of the Nervous System, 1882.

[4] Cathelineau, Arch. Gén. de Méd., April, 1894.

in them often rather cutaneous anæsthesia, with some trembling, than an obvious failure of co-ordinating power. It must also be remembered that the two diseases sometimes exist in combination.

With reference to the distinction of progressive locomotor ataxia from most of the *diseases of the spinal cord*, the extreme rarity of muscular spasm in ataxia must be dwelt on; from spinal paraplegia the result of myelitis it differs in the fact that the muscles act with strength, the patient can flex and extend his legs and kick vigorously, while in spinal myelitis the affected limbs cannot move, though the knee-jerk may be excessive. The lightning pains are not entirely to be trusted to in diagnosis, for they may happen in acute myelitis as well as in spinal pachymeningitis and in disseminated sclerosis. The absence of the knee-jerk in locomotor ataxia is of very great value. Its presence, in addition to the tremor, the nystagmus, and the scanning speech, distinguishes *disseminated cerebro-spinal sclerosis*. But mixed symptoms may exist from the different forms of sclerosis being combined. In *ataxic paraplegia* we have both disease of the posterior and lateral columns and a combination of the symptoms of spastic paraplegia and of locomotor ataxia. The knee-jerk is excessive, ankle-clonus is present, and there are extensor spasms in addition to weakness and to the incoördination; but no lightning pains or loss of light reflex attend the ataxia, as in tabes.

Putnam and Dana have described cases presenting *chronic sclerosis of the posterior* and *lateral columns*, especially of the pyramidal and cerebellar tracts, which are very puzzling. Among the symptoms are numbness in the extremities, progressive loss of strength, and wasting. The knee-jerks are at first exaggerated, but later they are enfeebled or lost, and paraplegia develops. The lower extremities suffer in greater degree than the upper. Mental symptoms may appear.

There is a chronic degeneration of the spinal cord having its chief seat in the posterior columns and the lateral pyramidal tracts, which mostly develops in childhood. It is often hereditary, usually occurs in families, is probably congenital in origin, and has as its chief symptom ataxia. This disease is known as *Friedreich's ataxia*, and also as *hereditary ataxia*, and is of very long duration. The disorder of coordination shows first in the lower extremities, and advances upward, at last affecting the organs of speech. The patellar tendon reflex is generally abolished; nystagmus and vertigo are frequent; while in the later stages spasms and contractions of muscles, curvature of the spine, want of control in keeping any part of the body quiet, and palsies, are not uncommon. Unlike what takes place in locomotor ataxia, we note no disorder of cutaneous sensibility, no

lancinating pains, no atrophy of the optic nerves, no Argyll-Robertson pupil, no trophic lesions, no visceral disturbances.[1]

From *diphtheritic paralysis* we distinguish tabes by the history of the malady, the absence of pain, and by the paralysis of accommodation and of the palate that precedes the muscular weakness. Loss of knee-jerk exists in both, and occasionally incoördination is met with in the former. In *multiple neuritis* this, too, may happen; but the marked muscular and nerve tenderness, the changed electric reactions, the normal pupils, the absence of the lightning pains, the more decided loss of muscular power, and, usually, the evidence of alcoholism, tell the true meaning.

A diminution or loss of the muscular sense—that guiding sense by which we judge of the position of the limbs, by which we are conscious of their movements—occasions difficulty in diagnosis, since in locomotor ataxia the muscular sense may be also deficient. On the other hand, in the former morbid state the motion may be somewhat impaired, for, as in tabes, the feet may feel numb in standing and in walking, and the patient be unable to walk in the dark. But there is this difference: where merely the muscular sense is affected, he can walk and perform all movements, even those of a complex nature, without vacillation, so long as his eye is fixed on them and superintends and gives them direction; while in tabes the derangement of muscular co-ordination renders, even with the aid of sight, the movements uncertain and irregular. Then cutaneous anæsthesia is apt to coexist with this malady. The treatment, too, will throw light on a doubtful case: the local use of electricity will usually cure the loss of muscular sense, as seen principally in hysterical paralysis; it has no curative effect in ataxia.

Irrespective of the affection of muscular sense, the greatest similarity to locomotor ataxia I have seen has been in several cases of *hysteria;* one in particular, in a very anæmic woman, resembled it closely; and it may be a question whether the nutrition of the parts affected in ataxia was not disordered, and the nervous structure functionally disturbed. I desire particularly to call attention to these cases, which can be distinguished by their history, the usual coexistence of anæmia, and the absence of severe darting pains. Yet pains may also happen in the hysterical complaint, as in a case I saw with Dr. Webb;[2] but this is uncommon. Moreover, the apparent want

[1] For an admirable analysis of cases, see Crozer Griffith's paper in the Transactions of the College of Physicians of Philadelphia, 1888. Sanger Brown, Chicago Medical Recorder, Feb. 1892, publishes a very striking family tree.

[2] Amer. Journ. Med. Sci., Jan. 1876.

of muscular co-ordination is more irregular in its manifestations, the knee-jerk is not lost,—though rigidity of the limbs may make this very difficult to ascertain,—and the cases recover. So, I think, may cases of locomotor ataxia due to special causes. For I have seen cases in *syphilitic* patients, typical in everything except perhaps the severity of the neuralgic pain, essentially typical in the muscular phenomena and in the inability to walk with closed eyes, in which a gradual and nearly complete recovery took place. Here the lesion was probably removed or greatly influenced by the anti-syphilitic treatment, and a true or extensive sclerotic degeneration of the affected parts did not take place.

Diseases of the Cerebellum.—Diseases of the cerebellum produce many of the phenomena regarded as peculiar to locomotor ataxia. But the gait of the patient is that of a drunken man: when attempting to walk, he leans to one side, moves in arcs of a circle, or describes zigzags; and when standing erect, his body swings backward and forward, or from side to side, though his feet remain quietly fixed on the ground. In ataxia, on the other hand, the muscular contractions in the erect position or during attempts at walking are strong and sudden, more like spasms, yet not spasmodic, and have as their object to keep the body in the line of gravity; and the walk, though accomplished with difficulty, is straight, not reeling; the affected person, too, while he is walking, does not take his eyes off the ground or off his feet, from fear of falling; but he is not giddy. The peculiar gait of cerebellar affections is particularly found when the middle lobe is involved. Disease spreading from the cerebellum gives rise to hypoglossal, facial, and other local palsies. In diseases of the cerebellum we find vertiginous sensations, especially during attempts at locomotion, which may be easier and straighter with the eyes shut than with them open; vomiting, particularly at the onset of the complaint, aggravated or brought on by the erect posture; nystagmus; severe headache, occipital or frontal, when the head is bent; defective vision, but with normal pupillary reaction, or double vision, though the eye-disturbances may or may not be associated with choked disk or optic neuritis; no diminution either of power of motion or of sensibility, unless from pressure on adjacent parts; and in some instances rotary movements and hemiplegia. Rotary movements are regarded as a special proof of affection of the cerebellar peduncles. The knee-jerks are sometimes wanting, sometimes exaggerated; there are no leg-pains. When the disease is localized in one hemisphere of the cerebellum, it may cause no symptoms.

Tremor.

Any involuntary agitation of the body, or of part of it, without marked muscular contraction or impediment to voluntary movement, is called tremor. The trembling depends upon a weakening of the muscular and nervous systems. It is common in old age, in convalescence from debilitating diseases, in hysteria, in neurasthenia, and during chills. We also find it in workers in mercury or in lead or in arsenic: in those who abuse alcoholic stimulants or coffee or tobacco, or who are addicted to the use of opium: and in cases of exophthalmic goitre. It may be connected with an organic disease of the nervous centres, as in cerebro-spinal sclerosis: and it constitutes the main symptom of the disorder known as paralysis agitans.

Tremor is easily recognized. Yet it may be confounded with muscular twitchings. But it differs from these spasmodic movements by being more incessant, and unconnected with decided muscular contractions. In nervous, susceptible persons laboring under an acute attack of disease, it is at times combined with great restlessness, and is apt to be mistaken for a convulsive state. Here again it may be distinguished by the absence of muscular contractions, and by the unintermitting irregular motions. Tremor, which is produced or at least exaggerated by voluntary motion, is known as *intention tremor*.

Paralysis Agitans.—Tremor is the chief symptom of paralysis agitans or shaking palsy. The trembling consists of fine small movements, is combined with muscular weakness, or rather with slowness of muscular action, and, while increased by exertion and mental excitement, it persists during rest, though it ceases during sleep. It usually follows continuous mental strain, emotional shock, prolonged exposure to damp, or some depressing acute affection in elderly persons: it may be due to trauma or to excessive use of a member; it comes on slowly and progresses slowly: it ordinarily begins in the hand or foot and gradually becomes general. The disease lasts for years: as it advances, the patient loses his equilibrium in walking, leans forward or walks on the fore part of the foot, and is rapidly propelled forward, but he may be propelled to one or the other side or even backward.

The trembling takes place over the entire body, and sometimes involves the head. It is in more or less continuous oscillations, at first, at least to a certain extent, controlled by the will. It is increased by emotional influences, and lessened by active or passive movement. The muscles react to both the faradic and the galvanic current. The expression of the countenance is vacant and fixed: the handwriting

is tremulous, the voice piping, monotonous, the speech indistinct, at times hurried; the muscles of the extremities become rigid, especially the flexors, producing deformities like those of rheumatoid arthritis. Sensation is unaffected, though there are abnormal subjective sensations and great restlessness. Complaints are made of cramps, of muscular stiffness, especially in the extremities, and of a sense of excessive heat, associated, indeed, with increased temperature of the surface and sweating. There are no cerebral symptoms; yet hypochondriasis and loss of intellectual power occur as the disease progresses. The hands are apt to assume a position as in writing. The knee-jerk is normal; it may be increased. In exceptional instances tremors are absent. There is growing belief that paralysis agitans is dependent upon nutritive change in the motor cells of the cortex of the brain.

Under the name of *simple senile paraplegia* Gowers has described an affection similar to paralysis agitans without tremor, in which this malady manifests itself only by stiff movement and by weakness of the limbs, face, and trunk. In simple senile paraplegia these signs show themselves especially in the legs, which gradually become weaker and weaker, but without there being wasting or sensory disturbance; there are also slight symptoms in the arms and face. The knee-jerk is normal, and foot-clonus is not observed. The disease is especially met with after fifty years of age. There are supposed to be degenerative changes in the leg-centres.

Multiple Cerebro-Spinal Sclerosis.—Different is the palsy dependent upon multiple or disseminated *cerebro-spinal sclerosis*, or *Charcot's disease*. The symptoms of this vary somewhat, as the nodules of hardened tissue affect the brain or the cord first. We have always tremor and paralysis, and sometimes sensory changes. The trembling may show itself from the start in the tongue or the eyeball, and with it we usually find headache, vertigo, failure of sight, nystagmus, amblyopia, impaired hearing, and at times gastralgia and vomiting. The want of power manifests itself in all the extremities, yet the lower exhibit the palsy most plainly, while the characteristic trembling is most evident in the arms; unlike paralysis agitans, the paresis or paralysis often precedes the tremor. Save in rare instances, the trembling, the most perfect example of intention tremor, is not witnessed except when the muscles are put into motion; stops, therefore, entirely or nearly so when they are at rest: it is usually tested by letting the patient pass a glass of water to his mouth. It occurs in decided jerks, and markedly affects the head, when this is moved at all. The gait is uncertain and tottering, and attempts at walking

increase the tremor. The voice is weak, the speech slow and scanning; there is mental enfeeblement, with failure of memory. Sensation is at first not affected, nor are the sphincters; but we may have hyperæsthesia, or anæsthesia, or paræsthesia, and girdle-pains. One or more cranial nerves may be involved in the sclerotic process, with resulting disturbance of function. The tendon reflexes are generally exaggerated, and foot-clonus is not uncommon. A peculiarity pointed out by Charcot is that the pupils move under light, and that the papilla is yellowish. Towards the end muscular cramps followed by contractions, and disorders of deglutition and of respiration, happen, or there may be attacks of an apoplectic character. It is in very advanced cases only that the electro-muscular contractility or the galvanic irritability of the nerves is decidedly diminished. Multiple sclerosis is most common between twenty-five and thirty-five, and lasts for years. One of its striking features is that long delusive periods of marked improvement occur.

The description given shows the dissimilarity between it and paralysis agitans. The most difficult diagnosis is as regards Friedreich's ataxia, when, as it occasionally does, disseminated sclerosis happens in the young. The disturbance of co-ordination in the former malady and the common loss of knee-jerk are the most obvious differences.

There is a form of disease in which the symptoms appear like those of disseminated sclerosis, and are yet due to an infectious process, such as scarlet fever, measles, variola, typhoid fever, and influenza. The tremor aggravated by intention, the scanning speech, the drooping lip, the dull expression of face and general air of stupidity, the spastic gait with exaggerated deep reflexes, are common to both. Nystagmus has, however, not been observed in this *pseudo-disseminated* sclerosis, and the cases recover. They are in their general character, except in the special symptoms that approximate them to sclerosis, like the pseudo-ataxia which is also observed after various acute infectious diseases.[1]

Hysteria may present symptoms resembling those of cerebro-spinal sclerosis, but marked and persistent ankle-clonus is wanting, as is also the plantar-reflex, while anæsthesia of the lower extremities is more likely to be present. In hysteria, difficulty in micturition is usual, a dissociated or instar a stress over increased frequency of micturition and scanty secretion.

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[1] See Westphal's paper in Arch. f. Psych. u. Nerv. v. 87; also cases collected by Dawson Williams, Med.-Chir. Trans. vol. xlvii. 1864.

There are other, though far less common, forms of tremor connected with organic disease, such as the *post-hemiplegic tremor* and the tremor in *spasmodic tabes*. In both the history of the case and the attending muscular disorder, with the violent but rhythmical tremors on attempted motion in the latter affection, are of great significance. As an organic tremor, too, may be classed that of old age. In this *senile tremor* the trembling is most probably due to degenerative changes in the motor tract. At first it happens only on voluntary movement, stopping during repose and sleep, though ultimately it continues during rest as well as during motion. It begins in the hands, but extends markedly to the neck and head, and finally becomes very much like the tremor of paralysis agitans. Dana,[1] studying this and other forms of tremor with great accuracy by means of Dudgeon's sphygmograph, states senile tremor, indeed, to be the evidence of an abortive form of paralysis agitans.

Functional Tremors.—There is a group of tremors in which there is no organic cause, or at least the cause is so fine as to elude detection. Toxic tremors belong to this group, and we will look at their characteristics.

Alcoholic tremor occurs only on movement. It is irregular, and of considerable range. It is very pronounced in the arms, face, and tongue; in the legs it generally shows itself only when they are put in action, as in an attempt to stand. It is associated, in acute cases especially, with great restlessness, and muscular twitchings are not uncommon. The trembling is usually worse in the morning. Then, too, in its diagnosis we lay stress on the habits of the patient.

Tobacco tremor is a fine tremor which more especially happens in the hands. It is sometimes seen in the tongue, which is smooth and shiny, and is apt to be combined with a relaxed skin, an irritable heart, and feebleness of sight.

Lead tremor is also a fine tremor. It is irregular in its distribution, usually seen in the hands, increased by motion, and not limited. It is often associated with beginning weakness of the extensor muscles of the forearm, with a blue line on the gums, and may involve the lips and tongue.

In *arsenical tremor* the trembling is wide-spread. There is also some difficulty in co-ordination, with beginning muscular paralysis, darting pains in the arms and legs, and diminution of tactile sensibility.

Mercurial tremor, another variety of tremor, usually appears first

[1] Medical News, Dec. 1892.

in the tongue and face, and later extends to the arms and legs. It is increased by emotion and effort, and is recognized by observing that the trembling and the incessant movements stop when the shaking limb is supported. Then the gradual manner in which the disease appears, its occurrence among persons whose occupations predispose them to the absorption of mercury, the wakefulness, the disorder of the digestive organs, and the sponginess of the gums, form a group of phenomena very characteristic.

Asthenic tremor, such as follows debilitating disease, is fine, is induced by voluntary movement, and is most marked after exertion or fatigue.

Hysterical tremor may be fine and irregular, or coarse and rhythmical. It is usually induced by emotion and movement, although the second variety may occur independently.

The tremor that commonly attends *exophthalmic goitre* may be regular, but is often coarse and jerky, and occurs only on movement.

There is a form of functional tremor which is found to be unconnected with any obvious cause and may last through life. This *essential tremor*, to call it by that name, comes on often in young persons and lasts through life. It is generally fine, but sometimes irregular and unequal, and is apt to be associated with other hysterical manifestations; it is sometimes very severe, as in the case recorded by Lloyd,[1] in which marked hysterical anorexia coexisted. It shows itself most markedly in the hands, is made worse by excitement and by attempts at motion, and to a great extent, but not entirely, ceases during rest. It is not associated with any other motor disturbance, and I have known it in persons of high intellectual endowments. It may not come on until middle age, is not dangerous, but is not curable. In an instance that came under my observation the father and the son, a young man, both had it at the same time to an equal degree. Kindred to it is the *hereditary tremor* described by Dana, which also is a fine tremor, that does not interfere with co-ordination, and which affects especially the upper extremities. It begins in infancy or childhood and continues during a lifetime, without shortening life. It is often brought out by an infectious fever, ceases during sleep, and may become associated with slight contractures of the fingers.[2]

[1] Amer. Journ. Med. Sci., Sept. 1893.

[2] Ibid., Oct. 1887.

Spasms—Convulsions.

Both these terms are applied to involuntary muscular contractions, with, perhaps, this difference: the word spasm is used when we wish to express the idea of less extensive muscular derangement, and especially when the muscles of organic life are believed to be involved; and convulsions, when the disorder affects the muscles of the whole body, or at least many muscles at once, and chiefly those of volition.

Spasms may be clonic or tonic. In *clonic* spasms the muscles are agitated by successive contractions and relaxations of their fibres. In *tonic* spasms the muscles are rigidly set, and retain for a time their contraction, in spite of every effort on our part, or on the part of the patient, to relax them. The most marked type of this disorder is seen in tetanus; the most perfect illustration of clonic spasms is furnished by hysteria.

Convulsions may be accompanied by a loss of consciousness, and abolished sensibility, as in epilepsy; or they may coexist with unclouded thought and unaltered sensibility, as in tetanus. What their immediate cause is, it is very difficult to determine. General evidence favors the cortex of the brain or the medulla as being the centres disturbed; but the irritation need not be direct, it may be reflected to them. Of their exciting cause we may say that, in those of susceptible nervous organizations, any extrinsic irritation, such as teething or disordered digestion, leads to a fit. Further causes are diseases of the brain; sudden interference with the circulation; profuse hemorrhages; anæmia; contaminated blood; the toxic influence of lead. Children often have convulsions as the precursors of febrile diseases. Convulsions have further been observed as a result of rupture of the stomach.[1] In point of diagnosis it is of great importance to distinguish whether their inroad is or is not symptomatic of a cerebral lesion. If there have been a previous manifestation of a brain affection, we may assume the convulsions to be the signal of cerebral mischief. Practically speaking, when convulsions are among the first signs of a malady, they are not apt to depend upon a disease of the brain; and even if recognized to form part of the symptoms of a cerebral lesion, we may conclude that the lesion has not reached its highest degree of development, but is still, as it were, irritative.

Besides separating convulsions or spasms in conformity with their centric or their eccentric origin, we must always attempt to ascertain

[1] O'Farrell, Lancet, vol. i., 1894, p. 1243.

the particular nature of the cause. If *centric*, is it congestion, inflammation, a tumor, sclerosis, or other lesion of the brain or membranes? or is it the convulsion due to influences the cognizance of which is not within our horizon? If *eccentric*, is it owing to an impure or impoverished blood, to retained poisons, to ptomaines, or is it peripheral from nerve lesion or intestinal or other visceral irritation? and in how far reflex? To solve these questions is often very difficult, and nothing but a careful analysis of all the phenomena of the case enables us even to approximate the truth.

Among the most extraordinary forms of spasm connected with increased reflex irritability of the cord is the so-called *saltatory spasm*, in which so violent a spasm of the legs takes place when the patient's feet touch the floor that he is thrown into the air. In some instances, as in one described by Bamberger, palpitation, dyspnœa, and inequality of the pupils also existed. Other forms of tonic or clonic spasm happen from reflex irritation of certain nerve-tracts, and these functional spasms produce for the time being the most singular contortions and deformities.

Rhythmic movements of the head, associated with nystagmus, are occasionally observed in infants and young children. The oscillation is sometimes horizontal, sometimes vertical, sometimes both. Many of these children are rhachitic, some are epileptic. Occasionally there is an antecedent history of traumatism. In some cases the condition is connected with defective light in the crowded dwellings of the poor, necessitating almost constant artificial illumination.[1]

Friedreich has described as "paramyoclonus multiplex" a condition of clonic spasm often recurring in paroxysms and involving the arms and legs and face and neck. The movements are increased by emotion, and may be controlled by voluntary effort.

Closely associated with spasms are other kinds of irregular muscular movements, such as cramps,—a contraction of short duration of one or of several muscles, occurring in paroxysms and attended with severe pain; rigidity,—a more lasting tonic contraction of the muscles; and the jerking movements of chorea. Now, some of these, especially localized spasm and even rigidity, have a strong connection with the seat and character of the lesion. Thus, broadly speaking, if we have spasm, perhaps alternating with chorea-like movements, confined to one arm, one leg, one group of muscles, we may infer an irritative lesion in the cortical motor area, affecting in this monospasm the centre presiding over the motion of the disordered

[1] Lewi. Medical News, Nov. 10, 1894.

parts. Early rigidity in the muscles, especially after hemorrhage, is apt to be associated with increased faradic and reflex excitability, but the contracted muscles become relaxed during sleep; in late rigidity the contraction or "contracture" is increased by movements, whether voluntary or passive.

DERANGED NUTRITION AND SECRETION.

Derangements of nutrition and secretion are especially manifest in paralyzed limbs or after nerve-wounds. But these obvious alterations need here only be referred to; it is the intention to speak rather of the less palpable phenomena, the trophoneuroses, in which, at first sight, the nervous system is not so distinctly concerned. For instance, there is to be noted the rapid development of blisters and bedsores in connection with marked cerebral and spinal lesions; the skin may become the seat of diverse eruptions, undergo modifications of color and structure, the secretions may be augmented or diminished, the muscles and joints show textural changes, swellings may happen affecting various portions of the body, either external or internal,—yet all be due to disturbed nervous influence, and the real disorder be in parts very different from where it appears. Then we find the trophic symptoms of atrophy of the muscles in acute poliomyelitis and in Friedreich's ataxia, in the latter affection often associated with blueness and coldness of the feet from vasomotor change.

To particularize with reference to a few of the derangements mentioned. There is the affection described as *herpes zoster*, in which the vesicles encircling half the circumference of the trunk are not a primary skin disorder, but the local expression of irritation of a nerve,—most generally of a dorso-intercostal neuralgia. Then we encounter instances of large vesicles or bullæ accompanying other neuralgias, as of the sciatic; and attacks of erythema having their origin in facial neuralgia. Furthermore, various kinds of spots and blotches, and thickenings of the periosteum and of the skin, have been noticed after this and other forms of neuralgia; and we have eruptions of zoster in chronic myelitis and rashes limited to the limbs affected with pain in locomotor ataxia; and eczema of nervous origin produced by reflex irritation in disorders of the urinary organs;[1] and ichthyosis of the lower extremities in chronic spinal diseases.

Oftentimes, too, these morbid appearances on the skin are combined with evidences of altered secretion. Thus, in a case related by Parrot,[2] in addition to the neuralgic paroxysms attended with san-

[1] Ord, St. Thomas's Hospital Reports, vol. vii., 1876.

[2] Gaz. Hebdom., 1859; Handfield Jones on Nervous Disorders.

guineous exudations at the painful parts, there occurred, at times, bloody sweating of the knees, thighs, hands, and face. Lachrymation was noticed in nearly half the cases of trigeminal neuralgia analyzed by Notta;[1] and one-sided furring of the tongue is a not uncommon phenomenon in this complaint. Associated with these evidences of altered secretion may be signs of altered nutrition, such as iritis, corneal clouding, and inflammation of the fascia or of the periosteum in contact with the aching nerve. Let us add that these manifestations of perverted nutrition are not confined to neuralgic disorders. Trophic changes occur also in diseases of the central nervous system. Thus, inflammatory affections of the joints have been observed to follow cerebral hemorrhages, and various spinal maladies, particularly acute myelitis; local dryness of the skin occurs in unilateral atrophy of the face, and in some cases of syringomyelia; a form of joint-mischief, of hydrarthrosis, has been specially described in locomotor ataxia by Charcot; affections of the joints have also been observed in syringomyelia; and the perforating ulcer of the foot has been found by Ball[2] and Fayard[3] to be often connected with locomotor ataxia. Perforating ulcer of the foot has, however, also been noticed in Morvan's disease.

Œdema happens also as a vasomotor change. Weir Mitchell[4] points out swelling of the limbs in menstrual periods. Furthermore, we find local œdematous swellings occurring in various parts of the body associated with intestinal disturbance, sometimes periodically and with an hereditary tendency, and this *angio-neurotic œdema* has been reported by Osler[5] as affecting members of a family for five generations.

Among the phenomena of altered secretion connected with nervous affections, one of the most striking is *excessive sweating*. In lesions of the cervical sympathetic on one side, we may have strictly unilateral sweating of the face and neck, the other side remaining perfectly dry;[6] and greater vascularity and increased temperature are concomitants. In lesions of the abdominal ganglia, profuse sweating also happens, and is apt to be combined with impeded secretion from the mucous coats of the bowels, as we at times find in instances of abdominal aneurism. Not that excessive sweating, whether localized

[1] Arch. Gén. de Méd., 1854.

[2] Trans. of Internat. Med. Congress, vol. ii., London, 1881.

[3] Thèse de Paris, 1881.

[4] Amer. Journ. Med. Sci., July, 1884.

[5] Ibid., April, 1888.

[6] As in the case recorded by W. Ogle, Med.-Chir. Trans., vol. lii.

or general, is always linked to an affection of the great sympathetic ganglia. We find local sweatings limited to the hands and feet without any signs of other disorder. And general sweatings, irrespective of those of colliquative character attending phthisis, or of those of malarial diseases, happen after low fevers, in influenza, in inactive states of the liver, and in some persons go on for years without obvious cause. It may be that in these cases the sympathetic system is really at fault, at least in so far that there is a reflex derangement of the vasomotor nerves, and of course, then, of the subcutaneous blood-vessels and of the glands they supply.

But these are not questions which we can here consider. Indeed, the *why* and the *how* of all these changes of secretion and nutrition attending nervous affections are still very uncertain.

To return to the clinical phenomena. Besides the external manifestations of altered secretion and nutrition, there are certain changes in internal organs, the expression of nervous derangement. There is, for instance, exophthalmic goitre; the pneumonia that results from injury to the vagus; the ophthalmia, which may even pass on to perforation of the cornea, that happens after paralysis of the trigeminus; the kidney disease which follows chronic spinal affections.

From the preceding pages it will have become apparent how many of the nervous complaints are functional, or are at least of necessity so regarded, though science is steadily narrowing their number. In consequence of the uncertainty respecting the functional affections, doubt is thrown over any anatomical or pathological classification of nervous diseases. I subjoin a table of the main affections, arranged according to their supposed sites. In several of the disorders regarded as functional modern research has indicated the probable organic cause. But from the point of view of the physician it would be premature to hold to a fixed lesion, and I contend rather for the classification being useful clinically than unimpeachable pathologically. Nor will it be adhered to in the description of nervous affections, which will be traced according to divisions formed by groups of symptoms and not in obedience to a pathological classification.

TABLE OF THE AFFECTIONS OF THE BRAIN AND SPINAL CORD.

CEREBRAL	*Organic*	Hyperæmia.
		Anæmia.
		Meningitis in its various forms.
		Hydrocephalus.
		Abscess.
		Softening.

TABLE OF THE AFFECTIONS OF THE BRAIN AND SPINAL CORD.—*Continued.*

CEREBRAL	*Organic*	Sclerosis.
		Hemorrhage (Apoplexy).
		Thrombosis.
		Embolism.
		Tumors, etc.
		Aneurism.
		Glosso-labio-laryngeal paralysis.
		Syphilitic affections.
	Functional	Delirium.
		Insanity (?).
		Hypochondriasis.
		Headache.
		Trance.
CEREBRO-SPINAL	*Organic*	Cerebro-spinal meningitis.
		Disseminated cerebro-spinal sclerosis.
		Paralysis agitans.
		Simple senile paraplegia.
		Hydrophobia.
		Tetanus.
	Functional	Occupation-neuroses.
		Epilepsy.
		Catalepsy.
		Ecstasy.
		Chorea.
		Hysteria.
		Neurasthenia.
SPINAL	*Organic*	Hyperæmia.
		Anæmia.
		Spinal meningitis.
		Myelitis in various forms.
		Softening.
		Atrophy.
		Sclerosis.
		Locomotor ataxia.
		Spastic paraplegia.
		Hereditary ataxia.
		Ataxic paraplegia.
		Spinal apoplexy.
		Tumors, etc.
		Syringomyelia.
		Syphilitic affections.
		Progressive muscular atrophy.
	Functional	Spinal irritation.
		Spinal exhaustion.
		Tremor.
		Reflex spasms due to irritation of the cord.
		Acute ascending paralysis.
		Myotonia.

Acute Affections of which Delirium is a Prominent Symptom.

This clinical group embraces the different forms of meningeal inflammation, delirium tremens, and acute mania.

Acute Meningitis.—By this term is understood an inflammation of the membranes of the brain, especially of the arachnoid and of the pia mater, or *acute leptomeningitis*. The dura mater is far less frequently attacked; very rarely, unless the morbid action be of syphilitic origin, or have extended from the bones of the cranium, or resulted from an injury.

The disease generally presents two well-marked stages. The first, or the stage of excitement, is characterized by intense headache, great restlessness, vomiting, a hard, frequent pulse, slow in proportion to the temperature, injected eye, often with a contracted pupil, strabismus, an increased sensibility to light and sound, obstinate constipation, irregular respiration, stiffness of the muscles of the neck, and soon by active delirium, and by convulsions. The temperature rarely exceeds 103° F. In the second stage the extremities are cold, the pupils dilated, the pulse is feeble and slower, and intermitting, or becomes extremely rapid and thread-like; involuntary passages occur; there is utter loss of mind and of sensibility,—in one word, coma or collapse. In this stage the temperature may fall below the normal, or may reach 106°. Not every case, however, has all these symptoms, or goes at once from the stage of excitement to that of collapse. There may be a well-defined period of transition, during which drowsiness appears. Again, the disease may be arrested before the signs of prostration are evident.

The attack may be preceded by sick stomach, buzzing in the ears, and vertigo, or it may set in with severe pain fixed to the forehead and increased by movement. In some cases it begins with delirium or convulsions. On the other hand, these signs may be absent.[1] Among the symptoms of the affection, even in the earliest stages, a persistent pain attacking one or both knees, violent, intensified on motion, unrelieved by local means, and connected neither with swelling nor with any other change in the form or appearance of the joint, has been particularly noticed.[2] Another sign, as of every form of meningitis, including the epidemic cerebro-spinal, is the so-called Kernig's sign,—an inability to extend the leg when the thigh is flexed at a right angle with the body.

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[1] In a paper by Church, in St. Bartholomew's Hospital Reports, vol. iv., several cases without delirium are narrated.

[2] Lund, quoted in Amer. Journ. Med. Sci., Oct. 1864.

The malady may pass rapidly through its stages, so rapidly that their distinctive features become confused and blended. Generally it does not last less, or much more, than a week. There is marked emaciation attending it.

Acute meningitis is brought on by alcoholism, by exposure, by depressing cares, by intense application to study, by a blow or fall upon the head, by disease of adjacent structures, or by syphilis; or it may occur in the course of chronic nephritis, of the wasting diseases of children, or of infectious processes, such as measles, scarlatina, smallpox, typhoid fever, and pyæmia, though it is rare under all these circumstances; finally, it may be due to pneumonia or to insolation. Bacteriologically it is chiefly owing to the meningococcus or to the pneumococcus meningitis, and this not only in connection with pneumonia, but as a separate malady. Meningitis sometimes affects mainly, or wholly, the coverings of the convex portion of the brain; at other times the inflammation is limited to the base. *Meningitis of the convexity* is not infrequently purulent, and, if purulent, temperatures of 104° to 105° are usual. It generally comes on suddenly, and is found to be connected with disease of the bones of the skull, with ear-disease, or to follow exposure to the rays of the sun. Severe headache, intense delirium, hyperæsthesia, spasms in the facial muscles of one side and in one or both arms, and hemiplegic weakness are among the most marked symptoms. According to Duchâtelet,[1] *meningitis of the base* may be discriminated by remissions in the delirium, and by the coexistence of spasmodic symptoms with profound and early coma. In some cases acute muscular pains with defective motor power, a clear mind until late in the disorder, a temperature of 105°, have been specially noticed.[2] Moreover, the longer duration of the malady, the delirium of varying intensity and later appearance, the intervals of clearness, and the late and incomplete palsies, are regarded as significant of this simple basilar meningitis.[3] Then persistent vomiting, paralysis of cranial nerves, marked rigidity of the neck, and early optic neuritis point to the base; optic neuritis is indeed rare in meningitis or meningo-encephalitis of the convexity. Yet there is no certainty in the diagnosis. Nor can we be sure of the membrane chiefly involved in the meningeal inflammation. Inflammation of the dura mater, or *pachymeningitis*, has the least severe and striking symptoms. It is most commonly noticed as

[1] Inflammation de l'Arachnoïde, p. 230.

[2] Dowse, Medical Times and Gazette, Feb. 1874.

[3] Huguenin, in Ziemssen's Cyclopædia.

due to extension from caries of the bone, to injuries of the head, to syphilis, or to sunstroke.

A form of inflammation of the cerebral meninges characterized by extravasation of blood between the dura mater and the pia-arachnoid is known as *hemorrhagic pachymeningitis*. It has been observed most commonly in the chronic insane, and in cases of chronic alcoholism. Among the symptoms to which this condition gives rise are apoplectiform seizures, headache, somnolence or coma, muscular weakness, nystagmus, smallness of the pupil, optic neuritis, headache, and vomiting.

Acute meningitis is not always easy of diagnosis. Leaving out for the present the other disorders belonging to the same group, such as acute mania and delirium tremens, it may be confounded with

Cerebritis;
Acute Softening;
Intracranial Tumor;
Ear Disease;
Head Symptoms of Continued Fevers;
Head Symptoms of Acute Rheumatism:
Head Symptoms of Acute Ulcerative Endocarditis;
Head Symptoms of Pneumonia; of Pericarditis.

Cerebritis.—There is little appreciable difference between acute inflammation of the brain-tissue and inflammation of the meninges. In truth, what we commonly call meningitis is not infrequently also cerebritis; since the diseased process extends readily from the tunics of the brain to the adjacent cerebral substance. We note acute cerebritis generally as the result of an injury to the head, of contiguous inflammation, of ear-disease, of septic influence, or of acute infective disease. We may suspect the brain-structure to have become involved if the sense of vision or of hearing be suddenly perverted; if the convulsions, the agitation of the limbs, and the tremors be very marked; if they occur chiefly upon one side; and if palsy of the limbs or face rapidly appears. The paralysis is generally hemiplegic. Where the palsies are limited, or the spasms or irregular choreic movements strictly unilateral, we may infer that the disease is limited, that we are dealing with an *acute focal encephalitis*. Where the brain structure is extensively involved, *diffuse encephalitis*, there is long-continued torpor of mind and body, and, in their valuable analysis of cases, Kneass and Brown[1] look upon this state of vacuity as of decided diagnostic value. The disease occurs especially in the young.

[1] Brain, vol. xvi., 1893.

Acute Softening.—The form of acute softening which simulates meningitis is that associated with delirium. Acute softening is almost always the result of arterial occlusion from embolism or thrombosis, or of venous thrombosis; arterial thrombosis is by far the most common cause. The existence of disease of the heart or of the blood-vessels, or of contracted kidney, gives us for the most part the clue to the case. The palsied side has often a decidedly higher temperature than the other side. The general temperature is that of fever, and may be high, 104° or more. In the cases of acute softening in very old persons, where an atheromatous state of the blood-vessels of the brain exists before the clogging, the rapid softening that may follow is apt to be preceded by restlessness, some mental confusion, and signs of a general breaking up of nerve-force; it is soon associated with disturbances of the bladder and rectum, and leads to coma. In the cases which I have seen there was neither much headache nor febrile disorder.

In rare cases there is a primary *acute hemorrhagic encephalitis* without obvious cause, though it is likely that the inflammation starts in the blood-vessels. In its main features it is similar to the acute encephalitis of children, where, however, the lesion is most apt to be cortical. In the primary acute encephalitis of adults punctiform hemorrhages are noticed.[1] The chief symptoms are rapidly developing coma and hemiplegia. The knee-jerks are preserved, though there may be crossed tendon reflexes in the lower extremities. A significant feature is the extraordinarily high temperature, especially before death. In the latter respect, it is like what is sometimes observed in recent hemiplegia following embolism or hemorrhage.

Intracranial Tumor.—A rapidly growing tumor or one of latent course may give rise to symptoms resembling those of meningitis, but the unchanged or steadily increasing paralysis, the marked optic neuritis, the progressive character of the symptoms, and the absence of febrile phenomena should suggest the cause.

Ear Disease.—Occasionally disease of the ear, with or without extension to the membranes or sinuses of the brain, may be attended with deceptive symptoms, the nature of which will become clear upon examination of the ear. In middle-ear disease severe headache, vomiting, high fever, delirium, convulsions, and retraction of the head indicate meningitis or abscess.

Head Symptoms of Continued Fevers.—In all the varieties of continued fever, but especially in typhoid and typhus, cerebral symptoms

[1] Strümpell, Deutsch. Arch. f. Klin. Med., Bd. xlvii., 1890, p. 53.

at times arise which bear a strong resemblance to those of meningitis, but without even traces of inflammation. How, then, are we to distinguish these fever cases from meningitis? or how ascertain if meningeal inflammation be really before us as a complication, as it sometimes is, of the fever? Unfortunately, there is no sign absolutely diagnostic. Cerebral auscultation affords us no help, for the blowing sound that is at times perceived is not constantly present in meningitis, and may be heard in health. As matters stand, a diagnosis can be established only by a close consideration of all the symptoms, and of the history, especially of the onset; by searching for the eruption of typhus or typhoid fever; by a careful study of the temperature curves; and by taking note of the expression of the countenance. The character of the delirium will be of service; it is ordinarily much more active when the membranes of the brain are inflamed, and is attended with throbbing of the arteries of the neck and face,—a symptom, however, not conclusive, for it may be noticed in low fevers,—and not infrequently with convulsions. The relation between headache and delirium may be of aid. In general diseases headache ceases when delirium sets in; in meningitis the two coexist. Then, too, we may lay stress on optic neuritis; on retraction of the head, if present; on the more intense headache; on the vomiting; and we may attach some, but not too great, importance to the red line made by drawing the nail across the forehead,—the meningitic streak. The most valuable differential sign is the loss of the knee-jerk, a loss that is apt to happen, at least temporarily, in meningitis.

Head Symptoms of Acute Rheumatism.—The morbid manifestations are like those of acute meningitis: restlessness, headache, and violent delirium, succeeded by coma; besides, rheumatic involvement of the muscles at the back of the neck may cause retraction of the head. The delirium is commonly of gradual approach, but it may come on suddenly. Generally it does not appear until the patient has been suffering for at least a week with acute rheumatism; and the sweats and swollen joints point out the malady with which it is combined. Examinations of the head, in cases which have proved rapidly fatal, fail to reveal, save in rare instances, any evidences of inflammatory action within the cranium. The abnormal signs are, as a rule, more properly attributable to the rheumatic poison seizing upon the brain, and to the altered condition of the blood. They are at times found to be connected with the setting in of inflammation of the membranes of the heart, or of pneumonia, or with albuminuria, or with plugs of

fibrin in the capillaries of the brain, and are frequently associated with a very high temperature.[1]

Head Symptoms of Acute Ulcerative Endocarditis.—The severe headache, the delirium, the somnolence, which may attend ulcerative endocarditis cause it to be confounded with meningitis. Generally, however, the fever is of a typhoid type: and the high temperature, the rigors, the marked swelling of the spleen, the absence of optic neuritis, are very significant, and so are the cardiac murmurs.

Head Symptoms of Pneumonia; of Pericarditis.—In both these maladies delirium may be met with of a character so active as to lead to the belief that the brain is involved in an inflammatory disease. The diagnosis is cleared up by a careful examination of the chest. Then we may lay stress on the violent delirium being unattended with spasmodic movements or with paralysis. The form of pneumonia which is mostly associated with delirium is inflammation of the upper lobes. True meningitis sometimes attends pneumonia, and is with great difficulty distinguished from the mere disturbance of the cerebral circulation just mentioned, unless persistent vomiting, and pressure on a cranial nerve, or optic neuritis show us the real meaning of the brain affection.

Tubercular Meningitis.—This is not a rare disease in children. It is a meningitis pre-eminently of the base, incited by the tubercle bacillus.

The premonitory signs of the malady are of great importance. The child has generally been ailing for some time; is restless, peevish, sleeps badly, complains of headache, and is troubled with a frequent, short cough, and with constipation. To these symptoms are soon added thirst, a slightly coated tongue, vomiting, a dry, feverish skin, an accelerated pulse, and grinding of the teeth, constituting the prominent features of the first stage of the affection. After four or five days the second stage is reached, and the brain symptoms become more clearly developed. The child shuns the light, puts the hand frequently to its head, and utters now and then a peculiar, sharp, distressing cry. At night the headache becomes worse, and is attended with fleeting delirium. A slight strabismus is observable, and the eyeballs oscillate. The pulse is very irregular in its rhythm, sometimes rapid and intermitting, then slow. The vomiting ceases,

[1] For a collection of cases, I may refer to a paper on Cerebral Rheumatism which I published in the American Journal of the Medical Sciences, Jan. 1875. Dr. Posner, in the German translation of this book, points out that the use of salicylic acid, now so much employed, may give rise to confusing cerebral symptoms, such as headache, vertigo, hallucinations, even delirium.

and there may be a remission in the symptoms, with restored intelligence; but the pulse remains irregular, the temperature is moderately elevated, the bowels are even more constipated than before, and the abdomen appears retracted. The third stage is one of complete stupor, accompanied or preceded by convulsions. The expression of the face is idiotic; the pupils are dilated; there is subsultus, and one side of the body is paralyzed. Deglutition is difficult; the surface is covered with cold sweats. This condition may last for days; repeated convulsions hasten its termination.

Can we distinguish the formidable complaint from *ordinary meningitis?* Seldom from meningitis of the base; generally from meningitis of the convexities. As regards the discrimination from the former malady, we are enabled to pronounce the affection to be tubercular meningitis, if we are familiar with the patient's antecedents, and are cognizant, previous to the seizure, of the presence of scrofula of bones or joints, or of tubercle in any of the internal organs, or are able at the time to detect scrofulous glands or tubercular phthisis. But without knowledge of this kind a positive diagnosis is impossible: we have nothing to direct us except the probability that the case is tubercular, because most instances of meningitis of the base are of that nature. This uncertainty does not exist with reference to the usual form of simple meningeal inflammation. We may generally distinguish the tubercular malady by its occurrence in an unhealthy person; by its insidious approach; by the absence of violent delirium; by the appearance of convulsions, not early, but late in the disease; by the far less violent headache, and the less degree of febrile excitement; by the notable remissions in several of the cerebral signs; by the chest symptoms, and the long duration of the affection. The ophthalmoscope gives no certain information; tubercles are not commonly found in the eye-ground, only optic neuritis or choked disks.

Tubercular meningitis is ordinarily attended with an effusion of serum into the ventricles, and it is plain that many of the symptoms are attributable to pressure of the fluid on portions of the brain. Now, how can we separate the malady, acute hydrocephalus as it used to be called, from dropsy of the brain or *chronic hydrocephalus?* Partly by the history of the case, and partly by the normal size of the head; for the water on the brain is not sufficient in amount nor is it there long enough to produce an appreciable augmentation of the cranium. Then, in chronic hydrocephalus the symptoms manifest themselves for years, from childhood even to adult life. The signs of a profound cerebral lesion appear gradually,

the special senses are by degrees enfeebled, but it is a long time before they are wholly abolished, or before complete loss of consciousness takes place.

As regards the diagnosis between tubercular meningitis and *acute hydrocephalus*, it need only be stated that the latter affection is in the vast majority of cases a synonym for the former. Yet we occasionally meet with instances in which acute hydrocephalus occurs unconnected with tubercle. It runs then either a latent course, or appears as an acute malady with symptoms similar to those of acute meningitis, a *serous meningitis*, beginning with fever or with convulsions, and often attended with marked choked disks, with intense restlessness, succeeded by drowsiness, and having periods of intermission of the symptoms and of apparent improvement; the pulse and temperature show great variations. Towards the end severe convulsions are common. The complaint, unlike tubercular meningitis, happens in previously healthy children, begins suddenly, and is of short duration. But the effusion may remain, and the disorder lead to chronic hydrocephalus.

There is a functional disturbance of the brain to discriminate from tubercular meningitis,—the *hydrocephaloid* disease described by Marshall Hall. It has a stage in which the little patient is restless and feverish, and a stage in which the countenance becomes pale, the breathing irregular, the voice husky, the pupils dilated and uninfluenced by light, and in which somnolence, coma, and even general convulsions occur. The symptoms indicate cerebral anæmia and nervous exhaustion. They generally come on after an enfeebling attack of illness, especially subsequent to protracted diarrhœa or loss of blood; sometimes they follow premature weaning. In the history of the case; in the less tendency to vomiting; in the irregularity of the pulse; in the flaccid and hollow state of the fontanel, so dissimilar to its prominent and tense condition in inflammation; and in the arrest of the threatening signs by stimulants and by tonics,—we find the guides which enable us to decide against the existence of an organic disease of the brain or its membranes.

But other affections besides those of the brain may be confounded with tubercular meningitis, such as typhoid fever and pneumonia. From *typhoid fever* tubercular meningitis may be distinguished by the frequent vomiting; by the retracted abdomen; by the constipation, except in instances of coexisting acute intestinal tuberculosis; by the normal size of the spleen; by the irregularity of the pulse; by the occurrence of convulsions and anæsthesia, and other signs of profound motor and sensory disturbance; by the lower heat, the thermometer

seldom rising above 102°; by the absence of the serum-reaction of the Widal test. I have never seen an eruption in tubercular meningitis; but Barthez and Rilliet speak of fugitive, imperfectly formed rose-spots being present in rare cases. The duration of the two complaints affords no help in diagnosis, since the one may last as long as the other.

Tubercular meningitis may be confounded with the *acute affections of the lungs*, especially acute pneumonia, which, in children especially, are not uncommonly associated with delirium and other brain symptoms. But the temperature is much higher; and a close examination of the chest reveals the cause of the disturbance of the brain. As regards acute phthisis the difficulty is sometimes great, for there may be in tubercular meningitis also signs of tubercular deposition in the lungs. The high temperature of acute tuberculosis and the course of the cerebral symptoms, should these be present, would alone be conclusive. As a point in the diagnosis of the tubercular meningitis of children, with reference to the attending chest symptoms, Gee[1] mentions that the chest heaves equally on both sides, yet over a very large part, or even the whole, of one side, no respiratory sound is heard.

Tubercular meningitis is not so rare in adults as has been supposed, and presents, as Seitz in his admirable monograph has shown, marked features of pain in the head and temperature variations,[2] exhibiting a fever of moderate type, with irregular remissions. The deposit of tubercle both in adults and in children may not be confined to the head. Indeed, the observations of Liorilli[3] teach that the spinal cord is frequently implicated.

The points of differential diagnosis of the tubercular meningitis of adults are much the same as with reference to the disease in childhood. Yet one disorder is more apt to be confounded with it,—*hysteria*. Indeed, in young women the onset of the malady may develop very misleading hysterical symptoms. But on close examination we find the traits of the cerebral malady,—the temperature record of the attending fever, the unequal pupils, the divergent strabismus, the optic neuritis, the trophic changes in the skin, the incontinence of urine, the local beginning of the convulsions.

Cerebro-Spinal Meningitis.—Now and then cases of meningitis are encountered in which the inflammation affects simultane-

[1] Reynolds's System of Medicine, vol. ii.

[2] Die Meningitis tuberculosa der Erwachsenen.

[3] Archives de Physiologie, 1870.

ously the membranes of the brain and of the spine, and in which the symptoms of the cerebral malady are found to be blended with severe pain along the vertebral column, with retraction of the head, with convulsions, with rigidity of the muscles, with perverted cutaneous sensibility,—in short, with the phenomena denoting spinal meningitis. But such sporadic cases are of rare occurrence. Generally cerebro-spinal meningitis is not met with save as an epidemic disease that belongs clearly to the group of fevers, with which it will be described. But here may be pointed out the extreme difficulty of recognition of the sporadic non-epidemic cases. The early retraction of the head, the eruption, the temperature record of cerebro-spinal fever, the bacteriological results of lumbar puncture, are the most valuable diagnostic signs. Pneumonia, so common in this, may, as some cases mentioned by Gowers prove, also happen in the sporadic malady.

Delirium Tremens.—The prominent trait of this complaint is delirium, associated with trembling and with sleeplessness. It occurs in intemperate persons; yet such is not always the case, for we may find an affection identical with mania a potu in those who are not intemperate in the ordinary acceptation of the word, but whose nervous system has been racked by persistent mental anxiety, or by the use of other than alcoholic stimulants. I have seen such cases from the constant taking of chloral and of paraldehyde; and they may be noticed in morphine-takers.

Generally, however, delirium tremens is brought on by the abuse of intoxicating liquors. It is a current belief, and one which has found much favor among habitual drinkers, that a diminution or a sudden discontinuance of the accustomed beverage is followed by an onset of delirium. This may happen; but it is generally the reverse; it is a long-continued and unusually severe debauch which terminates in an attack of mania.

Let us look a little more closely at the mental wandering. It is very rarely fierce; nor is the patient taken up wholly with his delusions. He pays a certain amount of attention to surrounding objects, answers, perhaps in a rambling manner, the questions put to him, but fancies that animals are running around on his bed or are crawling on the walls, and is thereby, or by some equally distressing illusion, kept in horror and in dread. Or he imagines himself to be engaged in his ordinary occupations, and gives minute directions as to what he wishes done; tries to get out of bed, yet is quite tractable when thwarted in his efforts. He is very restless, his hands are constantly moving, and his delirium, to use the graphic epithet of Watson,

is a busy one. With it are associated sleeplessness, a frequent, soft pulse, utter loss of appetite, a moist, coated tongue, and a clammy skin. The tremor is irregular, wide in range, affects particularly the arms, face, and tongue, and is only induced on attempted movement. There is often, besides, spontaneous muscular twitching. The temperature is usually elevated, though rarely to a considerable degree; it seldom reaches 103° F.

How are we to distinguish the malady from one to which it bears a certain resemblance,—*acute meningitis?* Taking clearly expressed examples of each, we find the following marks of distinction: the pulse is different; tense and hard in meningeal inflammation, it is yielding and soft in delirium tremens. The skin and tongue are dry and feverish in the former affection, moist in the latter. Then the characteristics of the delirium are dissimilar: and in the one disease the mental wandering is combined with severe headache, but not with tremors; in the other, with tremors, but not with headache.

Yet in actual practice the diagnosis is not always so easy as it might appear to be at first sight, and here and there we meet with cases presenting symptoms the exact meaning of which it is puzzling to determine. The difficulty is mainly occasioned by extreme cerebral congestion, or by inflammatory action, having been produced by the same exciting cause that has brought on delirium tremens. In this blending of two morbid states, the pulse is, or soon becomes, tenser than in pure mania a potu; the temperature is apt to be higher, and the irritability of the stomach more marked and more persistent. In some instances, convulsions, strabismus, and deep stupor—carefully to be distinguished from the sleep which often announces the termination of mania a potu—set all doubt at rest. But when these signs are not present, we have to judge of the mischief that is going on within the cranium chiefly by the activity of the fever and by the appearances of the eye-ground, by finding choking of the disks. Yet caution is necessary in accepting as evidence phenomena which may be of diverse origin: the marked fever may be the result of, what is very frequent in delirium tremens, an intercurrent or coexisting pneumonia, or of a pulmonary apoplexy, as in a case I have seen. Then, again, we must not overlook the fact that in instances of pneumonia of the apex a delirium very similar to that of mania a potu may happen.

There is another point connected with the diagnosis of the malady which it is necessary to mention, and chiefly for the purpose of calling attention to a common error. The fact that a person known to be of bad habits is affected with delirium is received as a sure indication that the mental delusions have been produced by the abuse of ardent

spirits. But they may be owing to other causes: to fever, to a visceral inflammation: to acute mania. To avoid being deceived, we must lay stress rather on the special character of the delirium, and on the symptoms with which it is combined, than on its mere presence. In other words, delirium in inebriates is not of necessity the fruit of intemperance. In discussing acute mania we shall return to this subject.

When delirium tremens ends fatally, death takes place from exhaustion. The fatal issue is occasionally brought on by an intercurrent inflammation, especially of the lung, or by disease of the kidneys and uræmia. Sometimes, after the subsidence of the urgent cerebral symptoms, the patient dies very unexpectedly, and there are no morbid appearances in the brain or its membranes to account for the abrupt extinction of life. In many instances of these sudden deaths, a large amount of serum is found in the ventricles, or in the subarachnoid spaces.

Acute Mania.—It would be out of place to attempt to give, in a work of this kind, a detailed account of any of the forms of insanity: but, in its acute variety especially, it resembles other affections of the nervous system so closely that it cannot be wholly passed over.

There are two disorders with which acute mania is chiefly liable to be confounded,—acute meningitis and delirium tremens: and we shall for our purposes best learn the manifestations of acute mania by contrasting it with these maladies.

From *acute meningitis* mania differs in these essential particulars: the premonitory symptoms of the former are headache, drowsiness, and often a sense of tingling and of numbness in the extremities; these signs are, however, soon succeeded by the severe headache, tense pulse, decided fever, and optical illusions of the developed disease. The premonitory symptoms of acute mania, on the other hand, have generally existed for a longer time before the marked outbreak; some singular change of manner or of mode of thought commonly precedes the first violent attack of insanity, except in those cases in which the overthrow of reason results from a sudden, great grief, or from a violent shock to the nervous system. Further, when the delusions have taken full possession of the mind, the patient attempts to act up to them, and his bodily strength enables him to do so. He has little if any fever; no spasms; his pupils are not contracted; his stomach is not irritable; he does not suffer from headache, or at least does not complain of his head. It is needless to point out how all this differs from acute inflammation of the brain.

There is but little difficulty in discriminating between typical cases

of *delirium tremens* and of acute mania. The anxious countenance, the alarm, the good-natured loquacity and restlessness of the patient, his moist skin, compressible pulse, and creamy tongue, are very different from the ravings and excitement, or the stubborn silence alternating with the wild hallucinations, of insanity. Yet there are cases in which it is not easy to tell if the delusions are really due to intemperance: cases of insanity excited by drink in persons predisposed to mania. A few days, however, ordinarily remove all uncertainty: the person who was thought to be merely delirious is seen to become frantic after an intermission of quiet, or, unlike what happens in mania a potu, to be still out of his mind after he has had several sound sleeps. In one instance I met with, in which much doubt existed as to the diagnosis, the patient solved the doubt by jumping out of bed after having been quietly sleeping for hours, and, in a state of wild excitement, knocking down the nurse who tried to prevent her from leaving the room. Furthermore, in *acute alcoholic mania* there is a strong tendency to homicide, while in *acute melancholia* induced by drink the tendency is to suicide.

Diseases marked by Sudden Loss of Consciousness and of Voluntary Motion.

The chief diseases of this class are apoplexy, sunstroke, and catalepsy. Epilepsy, too, might be here regarded; but it will be more convenient to consider it with the convulsive affections.

Apoplexy.—This is coma coming on rapidly, and occurring nearly always as a result of intracerebral hemorrhage. It may be, however, also caused by sudden arrest of the blood-supply to the brain, by concussion, by congestion, and by laceration of the brain. Disease of the blood-vessels and miliary aneurisms are very commonly present, or we observe the malady in affections in which hemorrhages are prone to happen, such as in purpura, in scurvy, in pernicious anæmia, in leukæmia.

The malady has sometimes no prodromata; but not unfrequently it is preceded by great depression of spirits, by attacks of loss of memory, by illusions, by vitiated perceptions, by vertigo, by odd sensations in the head, or by one-sided weakness or numbness. The seizure is generally sudden, and the coma quickly developed. The patient falls to the ground, bereft of all consciousness. In other instances, before he sinks into the comatose sleep, there will be more or less pain in the head, sickness at the stomach, heaviness and confusion of thought, or even slight convulsions. Again, we may have convulsions a prominent feature almost from the onset.

When, whatever the beginning, the attack has reached its height, it presents these well-known features: the patient lies as if in a deep sleep, breathing laboriously and noisily, and each snoring inspiration is slow, followed by a flapping of the cheeks in expiration. The pulse is slow, full, at times irregular; the carotids throb violently, and the increased pulsation is particularly noticed in large extravasations; there is difficulty of deglutition; the pupils are immovable, and either contracted or dilated; the eye is half open; there is conjugate deviation. All thought, all sensation, all volition is suspended; the limbs are motionless, flaccid, and, when lifted, fall passively and to all appearances lifeless to the ground. Occasionally their muscles are rigid; but generally reflex irritability, superficial and deep, is lost. When this returns it appears on the unparalyzed side first. In severe cases the insensibility becomes greater, the breathing very irregular and of the Cheyne-Stokes variety, and involuntary discharges take place from the bladder and rectum.

If the patient recover from the comatose state, he does so generally in a short time: in a few hours, unless the lesion be very great, the intellectual faculties begin to resume their sway, and all the functions of the body are slowly restored to their natural condition. Yet there is a palpable exception to this in the muscular system. Paralysis of one side is apt to remain. The urine may be increased in amount and it may contain albumin and even sugar.

The temperature variations in apoplexy may be turned to useful diagnostic account. The temperature of the body is at first somewhat lowered, but this is followed by a stationary normal period, and not unfrequently by a rapid rise, which again, as the patient recovers, is succeeded by a return to the natural body heat. In severe cases, where large hemorrhages take place, the temperature seldom rises, or only rises to fall with the recurrence of the fatal bleeding, yet some apoplectic lesions of the pons and the medulla are throughout attended with elevation of temperature. If the stationary period be short or absent, and the body heat rise therefore almost continuously after the primary depression, the prospects of recovery are gloomy. From Dana's elaborate study,[1] we know, indeed, that in fatal cases of apoplexy a rise of rectal temperature to from 100° to 102° F. occurs, as a rule, on the first day after the seizure. On the second day, if the case be not immediately fatal, the temperature falls a little, averaging 101° or 101.5°. As regards the surface temperature, it is noted that in instances of extensive intracranial hemorrhage the temperature upon

[1] Amer. Journ. Med. Sci., June, 1894.

the paralyzed side is somewhat higher than upon the sound side, while in cases of thrombosis or embolism there is no such difference.

Apoplexy is very apt to happen after dinner and during sleep, and is most common during sudden changes of temperature. Liddell has shown that attacks are more usual in the spring. In New York he found the mortality greatest at this time of year.[1] One attack of apoplexy is likely, sooner or later, to be followed by another; and the reason of this is, that the predisposing cause is usually of a persistent character,—an organic cardiac malady, especially hypertrophy; Bright's disease; degeneration of the cerebral arteries; disseminated sclerosis, or softening of the brain. It is likely that the extravasation of blood is generally due to the same immediate cause, —to rupture of miliary aneurisms on the minute diseased arteries.

Now, is there anything at the time of the apoplexy, or after its most urgent symptoms have passed away, by which we can recognize whether the pressure on the brain results from a clot, from vascular obstruction, or from turgescence of the cerebral vessels? And, again, do the morbid manifestations furnish any clue to the seat of the hemorrhage? With reference to the former question, all clinical experience forces us to admit that, in any of the states mentioned, the actual signs may be the same, and that we never can be quite certain of the non-existence of a clot. It is true that when the apoplectic symptoms abate rapidly; when thought, however confused, soon returns; when the limbs are not paralyzed, or are so but imperfectly and for a short time, we have strong reason for believing that congestion, only, lies at the root of the disturbance; that, in other words, the case is one of those called simple apoplexy. But it is never possible to give a positive opinion, since a clot near the periphery of the brain may occasion the same phenomena as those specified. Attacks of cerebral congestion with apoplectic symptoms happen in the general paralysis of the insane. The features of this point out their nature.

With regard to a rapid effusion of serum, the difficulty of distinction from hemorrhage is very great. In fact, the only differential signs which were formerly claimed for *serous apoplexy*—namely, pallor of face and feebleness of pulse—are common in large sanguineous effusions; and we find absolutely no sign that can be looked upon as conclusive. Most of the cases of so-called serous apoplexy are instances of Bright's disease with serous effusion into the brain, and the very existence of the disease is now, for the most part, denied.

The *seat* of the hemorrhage can be detected with more certainty

[1] Treatise on Apoplexy, New York, 1873.

than the cause of the cerebral pressure; it could be detected with greater certainty were it not that the extravasation so often takes place into an already diseased brain. The order of frequency in which hemorrhage occurs into various parts of the brain is as follows: central ganglia, cerebrum ovale, cortex, pons, cerebellum, medulla, crus cerebri. In the majority of instances the blood is effused into one of the corpora striata and the *internal capsule*, or at the same time into the optic thalami, and we find only one-sided paralysis. If the lesion be in both hemispheres, the palsy is on both sides of the body, although more complete on one side than on the other. Yet a double-sided palsy does not justify an absolute opinion that the extravasation of blood into the brain-substance is double-sided; it betokens also an extravasation into the ventricles. But *ventricular hemorrhage* is distinguished by profound coma and by tonic contraction of the muscles, or by tonic alternating with clonic spasms, and rigidity of the muscles either on one or on both sides occurs; the respiration is much embarrassed, and the breath-sounds are obscured by râles. It is common in the very young and in the old, and paralysis is frequently absent, though it may be general.[1] Ventricular hemorrhage is more often secondary than primary, the blood having torn its way into the cavity. Hemorrhage limited to the *thalamus* may give rise to no symptoms unless the internal capsule be damaged, when slight hemiplegia, hemianæsthesia, and hemianopsia, with mobile spasm and motor incoördination, are apt to show themselves.

Hemorrhage into the *corpora quadrigemina* presents most frequently this combination of symptoms: muscular incoördination, impairment of sight and alteration of the pupils. *Cerebellar hemorrhage* gives rise to very temporary loss of consciousness, to unsteadiness of movement, and to frequent vomiting; vision is not affected. In instances in which there is hemiplegia it may or may not be on the same side as the lesion. In hemorrhage into one-half of the *pons*, there is palsy of the extremities on one side, and of the face on the other.[2] The pupils are often contracted, though they may be dilated and inactive. Disturbance of respiration is common. In lesions of the pons, too, as in those of the medulla, we have high and rapidly rising temperature almost from the onset, and we find an exception to the rule that the lateral deviation of the eyes and head, a sign so commonly present in apoplexy, is towards the side of the brain affection.[3] Anæsthesia and double-

[1] Sanders, Amer. Journ. Med. Sci., July, 1881.

[2] Gubler, Gazette Hebdomadaire, 1858, 1859.

[3] Bastian, Paralysis from Brain Disease.

sided palsy are often met with, and initial convulsions are very common, and are sometimes limited to the legs. There is vomiting as well as hyperpyrexia. Hemorrhage into the *medulla* is almost always immediately fatal; should the patient survive, symptoms of bulbar paralysis will be present.

In *cortical* bleedings we are apt to have localized convulsions and but slight palsy. Extravasation into the *cerebrum ovale* gives rise to similar symptoms, if it occur just beneath the cortex. Hemorrhage limited to one *crus cerebri* causes paralysis of the extremities on the opposite side and of the third nerve on the same side as the lesion. Hemorrhage limited to the *arachnoid*, with the blood poured into the subarachnoid spaces, occasions ordinarily pain in the head, somnolency, and profound coma without paralysis, and without anæsthesia or slow pulse, but with relaxation of the muscles, and sometimes with convulsions; now and then the symptoms assume, to all appearance, a remittent course. It is a very fatal form of apoplexy, occurring chiefly in new-born children, and after injuries to the head, or from the giving way of a diseased and widened artery, or in consequence of a rupture of one of the sinuses of the dura mater.

When the effusion of blood takes place between the dura mater and the arachnoid, it is generally the ultimate result of an inflammation and of subsequent changes of the inner surface of the dura mater; and on close inquiry the precursory symptoms of a disease of the membrane may be traced, perhaps, by the constant and localized pain, and the nocturnal restlessness. But the symptoms of the *hæmorrhagic pachymeningitis* or *hæmatoma* are obscure. It happens generally after fifty years of age, in the decrepit, in the insane, or in those suffering from pernicious anæmia, scurvy, emphysema, whooping-cough, alcoholism, or after head injuries. When the cyst ruptures in the thickened membrane, which it may not do for years, the signs are those of an apoplectic condition, lasting for eight or ten days. Headache, vomiting, nystagmus, and optic neuritis are among the main symptoms.

Let us now inquire how the diagnosis of apoplexy can be determined, and how this condition may be distinguished from other states which produce rapid loss of consciousness, or sudden paralysis. Not to mention epilepsy,—the phenomena of which we shall farther on contrast with those of apoplexy, and shall observe to differ chiefly in the prominence of the convulsive seizures; or meningitis,—in which fever, headache, and other signs of an acute cerebral disease precede insensibility; or a tumor,—which, save in the rarest instances, leads only very gradually to a comatose condition: or sunstroke,—

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struggling, and the symptoms become suddenly much ameliorated after the inhalation of ammonia. In narcotic poisoning, especially if from opium, the pupils are much contracted, and we are likely to encounter a gradual intensification of the coma. The patient, however, unless death be close at hand, can be momentarily roused from his deep sleep; and his calm, slow breathing is unlike the stertor of apoplexy. But when the hemorrhage has taken place into the pons Varolii, the diagnosis is very difficult, especially if the bleeding be extensive, for then we are apt to have a contraction of both pupils, and the respiration may not be stertorous; nor is there always at first paralysis. A symptom of great diagnostic significance, too, is the occurrence of convulsions. Still, this may happen in opium poisoning, and is not very rare in children.

Nitrobenzole, which operates as a narcotic poison in vapor as well as in a liquid state, may, in rapidly fatal cases, produce coma, which may be mistaken for the insensibility of apoplexy. But the poison is detected by its strong smell, resembling that of bitter almonds.[1] Poisoning by drinking chloroform gives rise to many of the symptoms of apoplexy; it is discerned by the odor of the breath, by the quick and tumultuous heart action that accompanies the stertorous breathing, by the relaxation of the limbs, by the deathlike aspect of the face, by the widely dilated pupils, and by the complete general anæsthesia.[2] Chloral insensibility is often preceded by vertigo and pains in the legs and arms, and is attended with flushing of the face, injected conjunctiva, a weak intermittent heart; the pulse may, however, be slow and full. Hydrocyanic acid poisoning produces profound insensibility, often attended by convulsions, and by peculiar breathing,—short inspiration with labored, prolonged expiration. The breath has the characteristic odor of prussic acid.

Uræmia.—The strong point in the diagnosis is that the coma is preceded by convulsions; exceptional instances are few indeed. An examination of the urine conduces, of course, to certainty; but, for obvious reasons, it cannot always aid us at once. Moreover, albumin—not, however, in large amounts—may occur in the urine after an apoplectic stroke, and after convulsions not uræmic. Puffy eyelids and swollen ankles, coma not profound, peculiar stertor seeming to emanate from the mouth, and pupils normal or dilated are symptoms that belong to uræmic coma. Unilateral convulsions or loss of power are indicative of cerebral mischief, and tell against uræmia.

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[1] Taylor, Guy's Hospital Reports, vol. x., 3d Series.

[2] As in the case reported in L'Union Médicale, Oct. 1864.

Diabetic Coma.—Diabetic coma generally begins, not abruptly, but with somnolency which passes into coma; it is often preceded by great oppression, and is attended with a rapid, weak pulse, but not with hemiplegia or other local palsies. But the chief distinction is by the tests, farther on described, which show an acid intoxication.

Syncope—Asphyxia.—The loss of consciousness in either of these states is as striking as in apoplexy. But there is this decided difference: the suspension of thought and of volition in a fainting-fit is due to failure of the circulation: hence the pulse is hardly or not at all felt, instead of being full, as in apoplexy. Further, the pallor of the face, the quiet or sighing respiration, the well-preserved reflexes, and the short duration of the syncope mark plainly the one affection from the other. And with reference to asphyxia, the turgid and livid face, the bluish lip, the distressed and embarrassed breathing preceding the convulsions, and the loss of consciousness, show clearly that the disturbance affects primarily the lungs and not the brain.

Acute Softening.—This state is so closely connected with cerebral embolism or thrombosis that an appreciation of the history of the case, and the causes that lead to occlusion of the vessels, tells us the meaning of the cerebral symptoms. Rapid softening, too, at times happens around a clot. In acute softening the mental phenomena are always obvious, the mind is much more obtuse or impaired than it is after the shock of cerebral hemorrhage is over. Durand Fardel[1] regards as a significant sign of acute softening an increased secretion from the mouth and eye.

Sudden Extensive Paralysis without Coma.—This is not a trait of apoplexy, but rather of occlusion of the large vessels. Sudden extensive paralysis without coma is ordinarily owing to the breaking down of a softened brain, most apt to have followed this occlusion; but it may be due to hemorrhage into the spinal column. Palsy from this source, unlike that caused by cerebral hemorrhage, is almost invariably double-sided, is accompanied by severe spinal pain, and, if the extravasation have taken place into the spinal meninges, by tonic spasms, like those of tetanus.

Protracted Sleep.—While recovering from acute diseases, the sick often sleep profoundly and for a long time. Yet there is little likelihood of confounding this with the sleep of apoplexy; for the antecedent circumstances reveal the meaning of this restoration of nature. Sometimes, however, persons sink into a deep and prolonged slumber

[1] Maladies des Vieillards.

without any previous ailment. Medical literature furnishes a number of such instances. In one recorded by Cousins,[1] the tendency to somnolency lasted for years. The patient frequently slept three, and sometimes five, days at a time. When he awoke he was well. In a case which I saw with Dr. Weir Mitchell,[2] the slumberer was aroused out of her trance several times by the exciting influence of electricity; but this finally lost its effect, and she relapsed into a sleep from which she awoke no more. These cases may give the impression of apoplexy, yet they do not resemble it strictly. They are unlike it in the gentle, noiseless breathing; in the feeble pulse; in the occasional motion of the body; and in the protracted unconsciousness. Then generally the patient can be roused sufficiently to take food. Prolonged somnolence is also among the marked symptoms of cerebral syphilis.[3] In some instances the disorder shows itself in a constant tendency to fall asleep for brief periods at a time. One patient I had slept repeatedly during the day, while on her feet, about five minutes at a time. She could be awakened by strong efforts. The short duration of the spells of sleep, and the absence of evidences of hysteria usual in trances, distinguish these cases of *narcolepsy* from trance. The narcolepsy may be, however, associated with catalepsy, and there are cases of undoubtedly hysterical origin. Such a case probably was the one of the lethargic Irish fasting girl, who is said to have existed for nearly six weeks without food; the disorder was ushered in by hysterical fits.[4] The recurrence of the sleeping fits, their innocuousness, and the absence of tremor and of progressive emaciation and of enlargement of the cervical glands distinguish narcolepsy from the dangerous *sleeping sickness* of Africa.

Cerebral Hysteria.—The actual similitude and the points of contrast between this curious state and apoplexy may be learned from the following sketch:

A married lady, of remarkably impressionable and nervous disposition, had been for many months suffering from amenorrhœa and from sluggish action of the bowels. She had also a constant cough, dependent upon tubercles in one of the lungs. She had been in very bad health, but by the beneficial effects of a sea-voyage, her symp-

[1] Medical Times and Gazette, April, 1863. See also a somewhat similar case, New York Medical Journal, Dec. 1867.

[2] Described by him, Transactions of College of Physicians, of Philadelphia, 1856.

[3] See cases in Lecture XVI., Buzzard on Diseases of the Nervous System, 1882.

[4] Lancet, July 15, 1893. Another sleeping girl is mentioned, *ibid.*, July 29, 1893.

toms were much amended. She began to gain flesh, and to take exercise without fatigue. She was, however, troubled with headache, and with pain at the lower part of the abdomen. On one occasion in the evening I ordered her some cathartic medicine; and in the morning she was better than usual, and in the liveliest spirits. A few hours afterwards I was sent for, and found her insensible. She had complained of a sudden, sharp cramp near the umbilicus, and had then ceased to speak. She remained unconscious for about twelve hours; yet not wholly so, for every now and then she opened her eyelids, muttered a word or two, a pleasant smile flitted over her countenance, but she soon relapsed into deep slumber. Her thumbs were drawn inward; she had occasional convulsive movements; the breathing was rapid, but not noisy; the pulse feeble,—at first slow, then frequent; her eyes squinted in the most decided manner. Stimulants and antispasmodics were freely given, but without much benefit, for she recovered from her lethargy only with the setting in of the most violent paroxysmal pains in the abdomen, shooting down the thigh, and accompanied by contractions of the muscles and by exquisite local tenderness. The next day, without much abatement of the suffering, she was perfectly conscious; but still she had an internal squint,—nay, was totally blind, and remained so for two days. During this time a menstrual discharge began, which in part relieved the abdominal pain, but it was not fully relieved until after the passage of large fecal masses. It is needless to point out how this display of hysteria differed from apoplexy.

Aphasia.—The faculty of speech may be interfered with by various lesions of the brain and of the pons and the medulla. Of these, some cause only disturbance of articulation, while others derange the higher speech processes. From the first result difficult or defective articulation, dysarthria or anarthria; from the second, the group of phenomena included in the designation *aphasia*. Though this is really a mere symptom, it is so prominent as seemingly to constitute the disorder. By aphasia is meant loss of the faculty of expression of thought, in consequence of loss either of the faculty of speech, or of that of communicating thought by writing or by gestures. The patient may be deprived of the ability of expressing himself in one of these ways, or in all. The loss of speech is the most common, and is apt to be associated with a very decided impairment of memory and an enfeeblement of intelligence. The disorder may be temporary, lasting but a few hours or some days, or it may continue for months or years. During its course the affected person is incapable

of recalling words to give utterance to his ideas; or if he can recall the words to the mind, and thus think, he cannot express them.

Very often the patient has but a few words at his control; he says "yes" or "no" for everything; or he uses wrong words, knowing perhaps that they are wrong, and sometimes only those of a profane kind; or he confuses merely some syllables in the words he employs; or he may not be able to utter an intelligible expression. Yet, while in this condition, there is no defect in the tongue, or lips, or palate, to account for the inability to talk; the act of swallowing is easily performed; and even where the aphasia is complicated with hemiplegia, it is not difficult to discern that the imperfect articulation and thick speech that may attend the palsy are not the cause of the singular disturbance of expression; a disturbance which will mostly show itself not simply by the failure to utter words, but also by the inability to recollect them and write them down. Indeed, it is necessary to bear in mind that, while these states may coexist, they also may be present separately. Thus, there may be inability to express thought in speech,—motor aphasia or word-mutism. With this there is often associated inability to express thought in writing,—agraphia. Then, there may be inability to comprehend spoken language,—word-deafness; or written or printed language,—"alexia." Most patients understand perfectly well what is said to them; some can read to themselves; and, unless the general intelligence be perceptibly affected, they can express themselves by signs and gestures. In some cases there is rather loss of memory, and forgetfulness and confusion, and perhaps a consequent use of wrong words: but when prompted the word is at once spoken. Where the power of expression only is lost, but the perception of thought-symbols is still present, the term "motor aphasia" is used. Where the latter is lost, it is customary to speak of the affection as "sensory aphasia:" word-deafness and alexia are forms of this. Again, there are cases in which words and ideas remain, but in which the power of forming correct sentences is greatly impaired, or is lost. This has been named "akataphasia" or paraphasia.

Slips of the tongue are by no means always to be regarded as aphasia, for very often these have a local cause, such as a sore tongue or lip, or a sharp tooth fretting the tongue, producing unusual sensations in the mouth.[1]

Aphasia is dependent upon disease situated in the frontal convolutions, in the seat of articulate language in the posterior part of the

[1] Vol. St. Thomas's Hospital Reports, vol. iv.

third frontal convolution of the left side of the cerebrum. This explains why the hemiplegia which may accompany aphasia is almost invariably right-sided. But it may be left-sided, if the corresponding parts of the right hemisphere have become the main centre of speech, as happens not infrequently in left-handed persons.[1] It has been further shown that the disturbance will be in the cortical substance of the speech-centre, of the auditory centre, or of the visual centre, or in the association-fibres, according to the form of aphasia. With the first, motor aphasia especially results, with the others, sensory aphasia.

The function of speech is subserved through sensory and motor processes. The former have to do with the reception of impressions from without, principally through hearing and sight, and the centres for these are in right-handed persons respectively in the first temporal convolution and in the angular gyrus on the left side; the motor speech processes originate in Broca's convolution. In disease destroying the latter and causing motor aphasia, the patient understands, but can speak but few words or syllables; in disease affecting the sensory centres, the auditory and visual appreciation of words is impaired or lost. When the first left temporal convolution is the seat of lesion, "auditory aphasia" or "word-deafness," with loss of hearing in the opposite ear, results, and words are wrongly used, and speech fails to convey any ideas. The words are heard merely as sounds. The patient fails to recognize his mistakes in speech, and is unable to correctly repeat words spoken to him. Disease of the angular gyrus on the left side gives rise to "visual aphasia," or "word-blindness," or even to "mind-blindness," the patient being unable to recognize words or objects through vision. There thus result alexia and perhaps agraphia, together with loss of vision in the right half of the visual fields.

Aphasia may be due to functional as well as to organic disease. In cases of aphasia of short duration and without palsy, there is probably merely congestion; in protracted cases, and those in which we find persisting hemiplegia, a large clot, or softening, or abscess, is likely to be present; embolism of the middle cerebral artery on the left side is prone to be the cause in cases that are associated with valvular disease of the heart and that have come on suddenly. Thrombosis from enfeebled nutrition will explain some of the cases of aphasia noticed

[1] The speech-centre is not invariably situated on the left side in right-handed persons, nor on the right in left-handed persons, as cases reported by Wadham (St. George's Hosp. Rep., 1868, iv. 245), Dickinson (Bastian, Aphasia and other Speech-Defects, 1898, p. 90), and Collier (Lancet, March 25, 1899, p. 824) amply demonstrate.

during convalescence from grave acute maladies. That consequent upon congestions ends in more or less rapid recovery; in the other forms, usually, either no improvement follows, or only a very partial gain of words takes place. Occasionally we meet with aphasia in hysteria or in epilepsy, in acute infectious diseases, in toxæmias, and in uræmia, or we encounter aphasia intimately connected with a syphilitic cachexia, and dependent most probably upon disease of the arteries. Transitory aphasia has been observed in the course of pneumonia. The complication usually appears towards the second or third day of the disease, being ordinarily preceded by headache and vertigo, and sometimes by numbness and tingling on the right side of the body. It may set in abruptly, without loss of consciousness, or be preceded by an apoplectiform seizure. There may be, in addition, transient palsy of the right side of the body. The manifestation is thought to be due to the action of the toxic products of the disease process.[1]

Aphasia may become manifest subsequent to attacks of vertigo, or to a paralytic stroke preceded or not by the ordinary signs of an apoplectic fit. Under these circumstances the diagnosis cannot be definitely made until consciousness has returned, and we have an opportunity of examining the state of the mind, and of the tongue, and of the muscles concerned in articulation, remembering that if there be merely difficulty in articulation the case is not one of aphasia.

Sunstroke.—Persons exposed to the scorching rays of the sun in midsummer often become dizzy, and fall to the ground insensible: they have had a sunstroke. The attack either takes place while the patient is still exposed to the sun, or, in rarer instances, he reaches his home with a staggering gait and a suffused face, giddy, faint, suffering from a dull, oppressive pain in the head, having a constant desire to micturate, and after some hours becomes unconscious. However the onset, the insensibility which occurs is generally complete, although it may be so but for a few minutes. Associated with it are a frequent pulse, a skin harsh and warm and sometimes very hot on the forehead, shallow, noisy breathing, difficulty in swallowing, contracted or, more generally, dilated pupils, and relaxation of the limbs. Scanty urine, delirium, and convulsions, which may or may not depend on uræmia, are not uncommon.

When we contrast these symptoms with those of apoplexy, we find the following marks of distinction: the pulse is not slow and full, but frequent and often feeble; there is more difficulty in deglutition, but a less snoring respiration; the coma does not ordinarily remain as

[1] Chantemesse, Semaine Médicale, 1893, No. 73, p. 582.

complete for so great a length of time, for soon the patient may be, temporarily at least, roused from his deep sleep; and no hemiplegia, no paralysis, either of the limbs or of the cheek, occurs. The temperature of the body is very high, 104° to 109°, and not below the normal, as it is at first in apoplexy. The after-symptoms, too, are different: in cerebral hemorrhage, paralysis; in sunstroke, feebleness of movement, but no paralysis. In the former, no marked, persistent headache; in the latter, headache, more or less chronic, always aggravated by walking in the sun, and often for months accompanied by signs of an exhausted nervous system, and in some instances by epileptic convulsions.

The question with regard to the discrimination of these morbid states is one of great practical value, as on the conclusion arrived at depends our therapeutic action; and generally it is readily determined by paying attention to the variance in the symptoms mentioned. But it must be confessed that we sometimes meet with ambiguous cases,—cases in which the signs of nervous exhaustion produced by exposure to heat are blended with those of cerebral congestion or hemorrhage excited by the same cause, and in which, when they terminate fatally, the autopsy shows not simply a changed blood, or pulmonary congestion, but turgescence of the cerebral vessels, or an extravasation. It may also be difficult to distinguish between sunstroke and acute alcoholism, particularly because those who drink freely are very prone to the disease. The chief distinguishing trait is in the high temperature of sunstroke, and the normal or lowered temperature of alcoholism.

The remarks just made refer to the most common form of sunstroke,—that attended with more or less sudden loss of consciousness. But there are cases in which the abnormal manifestations come on gradually, and in which the patient at no time becomes insensible. The chief symptoms are intense headache, nausea, prostration, and inability to perform any work requiring sustained attention. All these signs appear after protracted exposure to the sun; and they mend but tardily. In truth, in the slowly developed disorder, the subsequent nervous exhaustion and the paroxysms of headache are often much more persistent than are the same phenomena when they follow what seems to be the more violent form of the malady. Among the sequelæ of these apparently incomplete attacks are irritability of the bladder, incontinence of urine, and irregular action of the heart. But nothing is as striking as the loss of mental and bodily energy.

The symptoms of "insolatio," or sunstroke, may be induced by prolonged atmospheric heat while the patient is in-doors and not exposed to the rays of the sun. Such cases of heat-stroke are known

to occur in India even at midnight. They may be preceded by a sense of extreme weariness, by inability to sleep, by loss of appetite, by constipation and frequent micturition, and by deficient perspiration; or the signs of exhaustion, followed by more or less complete insensibility, appear without distinct prodromes. Cases of the kind under consideration may or may not show an increased or high temperature; generally they do.

Then, again, we find cases of *heat exhaustion*, often seen in our hot summers, in which there is from the first great tendency to syncope; the skin is pale, cool, and moist, the temperature not increased, the pulse very feeble, the pupils dilated, and stimulants freely given rapidly relieve the urgent symptoms.

The nature of heat exhaustion, as of sunstroke, is obscure. The latter is held to be a fever which is dependent upon heat.[1] Certain it is that the heat-centres are very much disturbed in the affection. It has also been suggested that sunstroke is an infectious disease, due to micro-organisms. In occasional instances meningitis rather than sunstroke follows exposure to the sun, and we find the ordinary symptoms of meningeal inflammation.

Catalepsy.—This is a sudden suspension of thought, of sensibility, and of voluntary motion, during the continuance of which the muscles become rigid, although they retain the exact position they happen to be placed in. The uncommon complaint occurs in paroxysms, which may last but a few minutes or for several hours, and during which the most complete anæsthesia, not only of the skin, but also of the deeper tissues, may occur.[2] Often consciousness is lost, but it may be only obscured. Respiration is disordered, the circulation is feeble, reflex action is abolished, and the temperature is lowered. The disorder is met with mainly in females, especially in hysterical females, and may alternate with outbreaks of hysteria. But it may also exist in the male sex, and be in either hereditary. It has even been noticed as an epidemic in localities where there are many families closely connected by intermarriage.[3] Nervous exhaustion or sudden alarm predisposes to the seizures, which at times recur periodically and last from a few minutes to a few hours.

Catalepsy may be mistaken for apoplexy, or even for death. It differs from apoplexy by its frequent recurrence; and further, during an attack the eyes are wide open, the pupils, although dilated, are

[1] H. C. Wood, Thermic Fever, or Sunstroke.

[2] As in the case reported by Lasègue, Archives Générales de Médecine, tome i., 1864.

[3] Vogt, Schmidt's Jahrbücher, Bd. cxx. p. 301.

very susceptible to light, and there is an absence of stertorous breathing as well as of the characteristic relaxation of the muscles or of the paralysis of apoplexy,—for the limbs are outstretched, or held in every conceivable annoying or painful position; yet as soon as consciousness is restored their movement fully returns. The pulse is not retarded; on the contrary, although feeble, it becomes very frequent.

The perplexing affection varies from a kindred state, *ecstasy*, in this: in the latter the loss of consciousness is not complete; the patient is merely insensible to external objects, because he is intensely absorbed in some vision present to his imagination, or in the contemplation of some subject to him of all-engrossing interest. But he is not statue-like; on the contrary, his countenance is animated and earnest, and he talks, declaims, sings.

There is a curious form of the disorder, which Sir Thomas Watson describes. It is an imperfect kind of catalepsy, called *daymare*, the affected person being incapable of moving or speaking, yet cognizant of all that goes on. These seizures of temporary deprivation of muscular power, without unconsciousness, are thought to depend upon a diseased state of the blood-vessels of the brain.

Feigned catalepsy may be distinguished from the true disease by the muscles quickly showing signs of fatigue, which they do not in real catalepsy. A pressure-drum, Charcot[1] found, fixed at the extremity of the outstretched limb in a person who feigns, will in a few minutes, in place of the straight, regular line, show crooked, very undulating traces, and the same irregularity is seen in the tracings of the pneumograph applied to the chest.

Catalepsy may be artificially induced, as we know from the interesting experiments on hypnotism which of late years have been made. Catalepsies of particular groups of muscles, or *partial catalepsies*, can also be artificially excited.

In the rare condition known as *trance*, or *lethargy*, there exists a state resembling sleep, from which the person can be roused with difficulty, if at all. It is principally associated with hysteria, although it has been observed as a result of excessive mental application and after exhausting disease. The patient is usually pallid. The extremities are, as a rule, relaxed, although they may be rigid for a time, and there may even be convulsions. The eyelids are closed and the eyes turned upward and to one side; the pupils vary in size, but react to light. Reflex action is usually lowered. Respiration and

[1] Third volume of Clinical Lectures, 1889.

circulation are greatly enfeebled; the peripheral temperature is subnormal. An attack lasts from a few hours to weeks. Cases in which it occurred without any obvious previous ailment have been mentioned while discussing protracted sleep. In *narcolepsy* there occur sudden short periods of day sleep, from which, however, the individual can be roused. These may recur spontaneously, or be induced by peripheral impressions; at times we find the condition in diabetic or gouty patients.

The affection described as *African lethargy*, or *sleeping sickness*, attacks negroes, principally on the west coast of Africa, and is characterized by somnolence of progressive degree, usually leading to great emaciation and to a fatal termination. Among its marked symptoms are drooping of the upper eyelid, puffiness of face, muscular tremor, itching, papular eruptions, a feeling of coldness even when lying in the broiling sun, and enlargement of the cervical, parotid, and submaxillary glands. The disease is thought by some to be due to the presence in the blood of the Filaria sanguinis; by others to a lesion of the pituitary body. It resembles beriberi, but does not show the hyperæsthesia of muscles, the abolished knee-jerk, the muscular atrophy this presents.

Diseases marked by Convulsions or Spasms.

Epilepsy.—Epilepsy is a disease the chief manifestation of which consists in recurring attacks of sudden loss of consciousness, attended with convulsive movements. The patient falls to the ground, without thought, without feeling, without the power of voluntary motion. He utters often a short, piercing cry, then a fearful struggle begins. The legs are stiff, and turned inward; the head is tossed backward, or from side to side; the mouth is distorted, the lips are covered with foam; the arms are outstretched and rigid, or thrown about with great force; the eyelids are half closed; the teeth are ground together, and the tongue is thrust between them, and often severely bitten. The face is often pale at the outset, but with the continuance of the tonic spasm the aspect becomes cyanotic. In a short while the rigidity gives way to clonic convulsions and the whole body may be agitated by violent movements, which may involve one side in greater degree than the other, and during which sometimes urine is passed. Gradually the convulsive movements become less violent and cease altogether, and the patient passes into a deep sleep, from which he awakes fatigued and exhausted, and dull in intellect. But these symptoms disappear, and he returns to his normal state of health. The attack generally occupies only a few minutes. In some cases,

however, the patient scarcely emerges from one attack before he enters upon another. This condition is known as the *status epilepticus*, and it may be kept up for hours.

Yet every paroxysm does not present the same phenomena, or run the same definite course. In many the attack is preceded by strange sensations; by a peculiar train of thought; by retching; by the feeling of a puff of air ascending from the extremities to the head. This "aura epileptica" is, however, far from constant. Moreover, it may exist and hardly be perceived: it may be an unfelt irritation starting from some peripheral nerve in any part of the skin, or from some organ not deeply seated, as the testicle, and its point of departure may be detected by observing, during the fit, in what neighborhood the first, or the most violent, or the most prolonged contractions occur. In very rare instances sudden spasms of the face and chest occur, with arrest of respiration, and with a subsequent clonic convulsion, yet with so little unconsciousness that it remains doubtful whether it has happened at all.

Some seizures are very light,—a transient suspension of consciousness, a slight twitching of some of the muscles, a fixed gaze, perhaps a decided impression of vertigo, and all is over. These abortive fits, the *petit mal*, or minor attacks, are very apt to precede by some days a severe attack, or several of them may take the place of the more turbulent form of the disorder. And they, like the graver epileptic convulsion, may present strange irregularities. They may manifest themselves, for instance, only in bursts of unmeaning laughter;[1] or intellectual derangement replaces the ordinary convulsive attack;[2] or there is mental wandering, with disposition to commit acts of violence. The attacks of epilepsy which are chiefly characterized by vertigo are distinguished from all other forms of vertigo by the loss of consciousness, however slight, they also present, and by the absence of any giddiness in the intervals. In nocturnal epilepsy ecchymoses on the face, conjunctival extravasations, a severe headache on awakening, and a sore tongue, may indicate what has happened in the night.

The epileptic paroxysm does not always pass off without leaving some trace of the profound disturbance it has occasioned. It may be followed by hemiplegia. Whether this be due, as Hughlings Jackson[3] asserts, to exhaustion of the nerve-centres following the excessive discharge of nerve-force bringing about the convulsion,

[1] George Paget, British Medical Journal, Feb. 1859.

[2] Thorne, on Masked Epilepsy, St. Bartholomew's Hosp. Rep., vol. vi.

[3] After-Effects of Epileptic Discharges, West Riding Reports, 1876.

it is certain that the palsy is very transient. Another sequel of the attack is aphasia; another, loss of voice; another, abdominal tenderness. As regards palsy, however, we must remember that epileptic fits may follow hemiplegia due to a vascular lesion, so-called *post-hemiplegic epilepsy*.

In the intervals between the seizures the patient is not in reality well. His temper is irritable, and his mental faculties slowly but certainly deteriorate. The loss of memory, particularly, is very marked; and dementia is not an unusual complication of long continued epilepsy. In some epileptics there is much excitement or a curious mental state preceding the seizures, or a violent and dangerous mania may follow them. Again, as I have noted in common with several observers, a temporary albuminuria is not unfrequently met with at the termination of the paroxysm.

True epilepsy is probably owing to functional or nutritional changes in the cortex of the brain, giving rise to excessive activity of nerve-cells leading to periodic discharges of nerve-force. Its most potent cause certainly is hereditary predisposition. Convulsions due to reflex irritation, to organic brain disease, and to toxic blood-states may result in true epilepsy. It is thus that the malady originates in injuries of nerves, in diseases of the skin, of the stomach and intestines, and of the uterus, in the irritation of worms, or in consequence of congenital phimosis,[1] or of chronic nasal catarrh.[2] Now, it is very important to discriminate between true epilepsy and *convulsions of excentric origin*; and to arrive at a conclusion is possible only by a thorough examination of all the constitutional symptoms, and by ascertaining the starting-point and tracing the course of the aura. The cases in which the aura is interrupted and the paroxysm arrested by a ligature are well known. Nothnagel cites an instance in which the aura began with peculiar sensations in the stomach, and the attack was stopped by swallowing table-salt. Convulsions may further be symptomatic of a cerebral disorder,—such as a tumor, cysticercus lodged in the organ, a syphilitic affection of the membranes, or a disturbance of the brain produced by disease of the skull-cap,—or, as in any disease-process affecting the cortex of the brain; or it may be due to watery blood, or vitiated blood full of abnormal ingredients, as in diseases of the kidneys, acting injuriously on the nutrition of the cerebral tissue. During the paroxysm it is impossible to determine the character of the convulsions; but in the

[1] Ultimo, *Lancet*, Feb. 1887.

[2] Voss, *Archiv. f. Ohrenheilk.*, Bd. xxiii. Heft 3 und 4, *Centralbl. f. Nervenheilk.*, June, 1887.

interval we may often do so by close attention to the history of the case, and by noting whether the patient enjoys the usual health of epileptic subjects, or presents signs of a chronic cerebral disorder, especially steady headache, palsies of cranial nerves, optic neuritis, vomiting. Romberg tells us that where affections of the bones of the head lie at the root of the complaint, the fits are readily induced by pressure upon the skull. Convulsions are often found in connection with ear disease, and especially with purulent otitis.[1] In those who inherit syphilis idiopathic epilepsy may happen.

Limited convulsive seizures are connected with disease of special convolutions; and if we have a convulsion which is limited, either a tonic or a clonic spasm of a group of muscles, we may from this monospasm diagnosticate an irritative lesion in the motor centre presiding over the disturbed part, though in the hemisphere opposite to the spasm. The irritative lesion is usually a meningo-encephalitis. The spasm most frequently originates in the hand, but we may also find it limited to a group of muscles in the face, or in the leg. At first there is no loss of consciousness during the seizures, but as the spasms spread and become unilateral, consciousness is lost. Convulsions due to syphilitic diseases are, for the most part, of the kind just described, and are the chief form of the cortical or so-called *Jacksonian epilepsy*. In *masked epilepsy* there is often an epileptic vertigo, with loss of consciousness and with twitching of some muscles, but the patient does not fall.

Much has been said of the distinction between epilepsy and *convulsions*. Now, as regards the seizure itself, there is no appreciable difference: the only diversity consists in the recurrence of the attack after intervals of comparative health, and in the non-existence of any disturbance from which convulsions are likely to arise, such as reflex irritation, organic brain disease, or a toxic blood-state. In young children the diagnosis may be a difficult matter; but the fits of epilepsy, very rare in them, are distinguishable by the dulness of intellect, and the slow mental and bodily development, observable in the intervals.

The diseases which are most apt to be confounded with epilepsy are *hysteria* and *apoplexy*. The former—like all the rest of the group now under discussion, like chorea, like tetanus, like hydrophobia—is discriminated by the absence of that perfect suspension of consciousness that takes place in epileptic seizures; and there are other marks of distinction, to which we shall presently refer. In apoplexy, as in

[1] Ormerod, Brain, April, 1883.

epilepsy, we meet with loss of consciousness, sometimes with convulsions. But these are, on the whole, rare, and coma precedes and does not follow them, as happens in epilepsy. Then, stertorous breathing and a slow, full pulse are not observed in epilepsy. Epileptic patients bite their tongues; this does not occur in apoplexy. In epilepsy the paroxysm seldom lasts longer than from ten to fifteen minutes before consciousness returns and before the convulsions cease; in apoplexy the insensibility is of much longer duration. Epilepsy is not usually followed by paralysis; apoplexy is commonly.

There is sometimes a close resemblance between syncope and abortive epilepsy, *petit mal.* But they occur under widely different conditions; and the loss as well as the return of consciousness is less abrupt in the one than in the other.

Epilepsy is at times *feigned;* yet impostors cannot feign it completely. They may bite their tongue; they may imitate the stertor, the foam at the mouth, the convulsions, the thumb drawn inward towards the palm, the confused air on awakening; they may simulate, although they rarely do so, the indifference to pain; yet there is one feature of the real attack they cannot copy,—the insensibility of the iris. No matter how skilful the dissembler, his pupils must contract when exposed to a strong light, they must dilate when the stimulus is withdrawn. Unfortunately, there are several difficulties in making this test an absolute one. In the first place, the pupils, during a fit cannot be always readily observed. In the second place, not in every case of epilepsy are they perfectly immovable; in some, though sluggish, they react to light. Again, as proved by Keen, violent muscular motion instantly dilates the pupil, and so long as the movement continues, so long will the iris act sluggishly, even when exposed to a bright light. Thus, muscular spasms alone, even when simulated, may cause the pupils to be dilated and inactive. A test more generally useful is the administration of ether. When given to an epileptic, its first effect is to increase the violence of the spasm, but eventually the patient passes into the deep sleep produced by ether, without any of the prior cerebral excitement; while in the malingerer this manifests itself by talking and laughing,—in fact, in the usual way.[1]

Chorea. This spasmodic affection is chiefly met with in young persons, especially in girls approaching the age of puberty. It is characterized by irregular clonic spasms of groups of muscles under the influence of the will, and mainly of those on one side of the body, together with muscular incoördination. But the patient is not

[1] Keen, Mitchell, and Morehouse, Amer. Journ. Med. Sci., Oct. 1864.

deprived of consciousness and of all power of voluntary motion. He knows what he is about, and can in part execute the movements he undertakes; yet his limbs are not completely under his control. They obey only his general directions, but not entirely, or at once: for the muscles jerk and pull as seem to them best, taking no heed of the time or the manner in which the will wishes any movement executed. In some cases the muscles of deglutition, of respiration, and of articulation become implicated; and difficulty in swallowing, in breathing, and in speaking occurs. A dilated pupil, too, acting sluggishly in response to light, may be met with among the phenomena of the malady. Often there are mental irritability and dulness of intellect. The urine contains urea and phosphates in excess.

Chorea is essentially a functional disorder of the nervous centres, especially of the cells of the cerebral cortex. In a large number of persons the malady is called into existence by an irritation of peripheral portions of the nervous system. Thus, a blow, a wound of a nerve, disorders of the uterus, painful menstruation, pregnancy, eye-strain, or gastric or intestinal affections may act as the exciting cause of the perverted muscular movements. In cases due to organic causes, endocarditis or plugging of the vessels leading to the corpus striatum is a common lesion, one-sided embolism giving rise to one-sided chorea.[1] The association with vegetations on the valves is in fatal cases certainly very frequent.[2] It has, indeed, been suggested that the wild, maniacal delirium, with subsequent rapid emaciation, which we meet with in some instances of chorea, has its origin in embolism.[3]

Chorea may be produced by strong mental emotion, especially by fright. It may follow scarlet fever, but it is more often the sequence of rheumatic fever or arises from the same diathesis that attends or occasions rheumatism. Yet this is not, as some have alleged, its only cause; for in a number of persons affected with chorea we fail to detect any proof of a rheumatic diathesis. Still, the greatest prevalence of chorea in the spring of the year coincides, as Morris J. Lewis[4] has shown, with the greatest prevalence of rheumatism. The action of special toxic substances on the brain is held by several to be the ultimate cause. As regards the cardiac complication, the presence of which is chiefly deduced from the existence of a murmur, the inference drawn of this being due to organic disease,—to endocarditis,—

[1] Hughlings Jackson, London Hospital Reports, vol. ii., and Edinburgh Medical Journal, Oct. 1868.

[2] Ogle, British and Foreign Medico-Chirurgical Review, 1868.

[3] Tuckwell, *ibid.*, Oct. 1867.

[4] Amer. Journ. Med. Sci., Sept. 1892, p. 251.

is not always accurate; for the murmur is often owing to anæmia, or dependent upon spasmodic action of the papillary muscles,—the same spasmodic action that is seen in the striated muscles of the face and of the extremities.

The disease is rarely fatal: but its duration is very variable; for, although it may be acute, lasting for six weeks or more, it may continue for months, even for years, and relapses are frequent. There are in chronic cases no attending cerebral symptoms, yet the mental faculties are not in a perfectly healthy state. The intellect of a choreic child develops slowly, and is enfeebled while the disorder lasts. In some cases paralysis supervenes; but it is not permanent, nor, indeed, of long duration. But those who have been choreic remain subject to nervous disorders; and I have known several instances in which the complaint has been, in after years, followed by epilepsy.

A chronic progressive form of chorea sometimes develops late in life, with, in many instances, a history of hereditary transmission. The movements in this *Huntington's chorea* usually appear first in the face and upper extremities, and gradually extend, and there is disturbance of speech. In the hereditary cases mental changes are common and sometimes pronounced, and gradual dementia is observed. In some parts of Italy there has been noticed an affection characterized by sudden, shock-like muscular contractions, with progressive palsy and wasting, and known as *electrical chorea*. Of its nature and cause we have no definite knowledge.

So-called *hysterical chorea* consists in general spasmodic movements occurring in hysterical subjects, but the movements are far more regular and rhythmical than those of true chorea, and are usually deliberate and of wide range.

The diagnosis of chorea is generally easy. The peculiar habit some children or even older persons get into of winking, or jerking the head, or of making other strange movements, the "habit-chorea" or "habit-spasm," as it has been called, is really a form of spasmodic tic, and is distinguished by its gradual development and its limitation to a single muscle, or group of muscles, or of associated muscles. This habit-spasm is not infrequently of reflex origin, as from the teeth or from eye-strain, but cases occur to which no cause can be assigned. Chorea with loss of power on one side, "paralytic chorea," is recognized in children by the occasional choreic movements, and by the loss of power which happens gradually.

Chorea from eye-strain is, as a ready test, discriminated by using atropine. Dr. Hansell employed this in many cases at my clinic with quick results. Atropine paralyzes the ciliary muscle; no effort of

accommodation can then be made; therefore muscular twitching, as well as headache or other functional disturbances from disordered accommodation, must cease after an interval of time long enough to break up the habit; chorea from constitutional causes will, of course, be unaffected by atropine or other paralysis of the ciliary muscle.

Chorea differs from the *spasms of acute cerebral disease* by the absence of fever, of delirium, and of coma, though we must bear in mind that we sometimes have elevation of temperature and mania in the chorea of pregnancy; from *epilepsy*, by its being continuous, by the non-existence of unconsciousness, and by the rarity with which the muscles jerk at a time when epileptic convulsions are frequent,—at night; from *tetanus* it is chiefly distinguished by not exhibiting tonic spasm. *Paralysis agitans* is, like chorea, attended with disturbed muscular movements. But we find weakness of the muscles and persistent tremor rather than spasmodic contraction and want of control over muscular motion. Then the history of the case, and the signs of general decay associated with the trembling, clearly distinguish paralysis agitans. In *cerebro-spinal sclerosis*, the scanning speech, the increased patellar tendon-reflex, the nystagmus, the occurrence of the jerks only when the muscles are put into motion, are most significant. Both affections, too, are encountered in persons older than are generally subject to chorea; especially in paralysis agitans. Multiple sclerosis happens, however, also in children, and we meet with cases of paralysis agitans affiliated to chorea; like it, too, originating in fright. But they differ in the motions repeating themselves rhythmically and symmetrically on the two sides of the body,[1] and in presenting nothing of the irregular and rapidly changing character of the true choreic movements.

Convulsive tremor, a paroxysmal affection in which severe muscular tremor arises several times in a day, differs from chorea in not being continuous, as it occurs in attacks lasting from fifteen to twenty minutes, and passing off gradually. The unrestrainable tremor affects the face, the arms, and the trunk, but not the lower extremities, and is associated with increased sensibility of the skin of the disturbed parts. Clonic spasms occurring as sudden contractions or shocks, and affecting pre-eminently the upper part of the limbs, have been delineated by Friedreich as *myoclonus multiplex;* the muscular irritability is much increased.

In *athetosis*, the condition described by Hammond, there is constantly recurring mobile spasm of the fingers and toes, with inability

[1] As in the case recorded by Sanders, Edin. Med. Journ., May, 1865.

to retain them in any position in which they may have been placed. Great tendency to distortion exists in the spasm, and we find, on the whole, much resemblance to localized chorea. But headache, vertigo, slowness of speech and of thought, numbness of the affected side, and pains in the parts which are the seat of mobile spasms, give us a very different clinical picture from chorea. During the spasm the fingers may be spread wide apart, giving the hand a characteristic appearance. Athetosis is most common in hemiplegics, especially in the cerebral hemiplegia of childhood, and coexists with contractures. It is supposed to be due to disease of a cortical motor centre. It has been observed to be bilateral in idiotic children. Similar to it is the mobile spasm that may be noticed in palsied limbs, the *post-hemiplegic chorea*. But here there is an admixture of tremulous movements.

Facial spasm differs from the spasmodic contractions of chorea in being always of equal intensity, and in the grimaces being strictly confined to the face, manifesting themselves in the same group of muscles, and generally existing only on one side of the face. Many cases of facial spasm are due to errors of refraction; in others it is the result of cortical disturbance or disease, or due to irritation of the filaments of the fifth nerve in the lachrymo-nasal canal. The spasm may be also of remote reflex origin, as from disorders of the uterus. There are also cases apparently idiopathic. In *convulsive tic*, as described by the French, the facial spasm is combined with signs of hysteria and with mental changes.

Writers' cramp, an affection in which every attempt at writing at once produces spasmodic action of the muscles of those fingers that are brought into play, is separated from chorea by its occurrence in individuals who have strained their muscles in using a pen continuously and rapidly; by the almost instant cessation of the spasm when the afflicted person ceases to write; and by the ease with which the fingers perform other motions and are capable of being used for every purpose except the one which has brought on the disorder. Pain, limited to the affected part, or more extensive, often attends this affection, at times induced only by writing, at other times spontaneous. There may also be weakness with or without spasm, and tremor, and local vasomotor manifestations, such as glossiness or turgid discoloration of the skin and undue heat and sweating. An analogous complaint, an "occupation neurosis," too, is encountered in seamstresses; also in telegraph-operators, particularly those who use the Morse instrument. These cramps, and all those of a similar kind caused by the occupation, such as in piano-players, in violinists, and in type-writers, car-drivers, stone-masons, cigarette-makers, shoe-

fitters,[1] have the same diagnostic sign that has just been mentioned as charcteristic of writers' cramp,—namely, that the spasm befalls only those muscles the overstrain of which has led to the affection, and that it ceases when the fatigued muscles are kept at rest or are brought into action for a different purpose. A form of cramp like that of writers' cramp happens in those engaged in preparing photographic plates;[2] and I have seen it in turners, engaged in what is called "oval turning."

There is a disorder, closely allied to chorea, which consists in repeated violent bobbings of the head, lasting many minutes at a time. These *salaam convulsions*, as Sir Charles Clarke called them, are a very obstinate complaint. They are most commonly met with in children, but have been known to occur in adults[3] and to lead frequently to impairment of the intellect.[4]

From *tetany* chorea differs in the spasm of the former being intermittent, remittent, or continuous and tonic, and not constant and clonic. The nodding movements of the head, sometimes lateral, sometimes rotary, with nystagmus, observed in *rhachitic* and ill-nourished infants are unlike those of chorea.

Hysteria.—Hysteria manifests itself for the most part in two forms, in convulsive paroxysms, or in local hysterical disorders. The description of hysteria here will deal chiefly with the symptoms of an hysterical paroxysm. Most of the local hysterical affections have been, or will be, considered in connection with the diseases they ape; and to attempt to scrutinize or to interpret connectedly all the false and contradictory signals this perplexing malady hangs out, is, in a work of this kind, manifestly impossible.

An hysterical fit may set in suddenly, under the influence of some violent mental emotion; but more generally it is preceded by altered spirits, by a sensation of pressure and of constriction at the pit of the stomach, which feeling ascends to the throat, and is likened by the patient to the rising of a ball. She becomes much agitated, sobs, laughs, her muscles contract violently, or she lies motionless, and apparently without the power of motion, until her seeming insensibility is disturbed by something she disapproves of, or fears. The heart palpitates; the breathing is often accelerated, irregular, and heaving; the pupils are dilated, their reflex gone.[5]

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[1] Moyer, Medical News, Feb. 1893.

[2] Napias, Gazette Médicale de Paris, No. 40, 1883.

[3] Levick, Amer. Journ. Med. Sci., Jan. 1862.

[4] Henry Barnes, Liverpool and Manchester Hospital Reports, 1873.

[5] Karplus, Wiener Med. Wochens., No. 52, 1896.

These hytserical outbursts differ from the spasms of *chorea* by the remissions, the patient remaining at times for months free from the convulsive movements. Moreover, there is not even partial or apparent unconsciousness in chorea. It is true that this malady and hysteria are sometimes combined, or rather that chorea happens in hysterical subjects, and is then brought about by imitation, and is apt to come on suddenly; yet it is remarkable how rarely fits of hysteria take place in those affected with chorea.

It is sometimes very difficult to distinguish between paroxysms of hysteria and of *epilepsy;* and it becomes the more difficult if the epileptic seizures occur in hysterical patients. Yet there are ordinarily many well-marked points of distinction between the two maladies, as will be seen from this table:

Epilepsy.	Hysteria.
Usually occurs without exciting cause.	Often induced by emotion.
Sets in with a scream.	Noisy during attack.
Sudden and complete loss of consciousness.	Gradual and only partial or apparent unconsciousness.
Livid face; escape of frothy saliva from the mouth; eyelids half open; eyeballs rolling; grinding of the teeth; biting of the tongue; more or less insensibility of the pupils to light.	Face flushed, or complexion unaltered; no froth on lips; eyelids closed; eyeballs fixed; neither grinding of the teeth nor biting of the tongue; pupils react readily.
Distortion of countenance.	No distortion of countenance.
Patient evinces no feeling.	Patient sighs, or laughs, or sobs.
Aura epileptica.	Globus hystericus.
Convulsions often more marked on one side than on the other; and at first tonic rather than clonic.	No such difference; convulsions tonic, followed by clonic.
Movements unlike voluntary acts.	Movements resemble voluntary acts.
May pass urine involuntarily.	Copious diuresis afterwards.
Paroxysm generally of short duration.	Paroxysm generally of longer duration.
Paroxysm followed by a heavy, half-comatose sleep, by headache, and by dulness of intellect.	Paroxysm not followed specially by sleep; patient often, after attack terminates, wakeful and depressed in spirits.
Frequently occurs at night.	Rarely occurs at night.
No particular connection with uterine or ovarian disturbance, although a paroxysm often takes place at the menstrual period.	Often connected with disorders of the uterus or ovaries, or of menstruation.

There are, however, spasms that occur in hysterical patients which, though a functional nervous affection, appear like a blending

of hysteria and epilepsy. Charcot[1] particularly has called attention to this *hystero-epilepsy*, and describes its distinctive traits as consisting in premonitory symptoms of rather long duration, and exhibiting an aura which, starting in most cases from the ovarian region, advances progressively to the head. The cry is prolonged and modulated, not short like the epileptic cry. The convulsions are identical; but, instead of entering subsequently upon a stage of snoring, the hystero-epileptic sobs, laughs, gesticulates violently, or is delirious and subject to hallucinations. In the ovarian form of hystero-epilepsy, pressure upon the ovary will invariably modify the symptoms, if not completely arrest the attack; whereas in epilepsy no such effect is produced. In the cases of hystero-epilepsy with repeated attacks, the temperature scarcely rises above the normal, as it rapidly does under similar circumstances in epilepsy. There is no epileptic vertigo; there are no abortive fits. The malady is not rarely observed in men and in children.

Hysteria is a psychoneurosis, and not an affection merely of paroxysms. In the intervals between them—there may be no paroxysms at all—we find peculiar and significant manifestations which should be understood, lest they be taken as the signs of other maladies. We observe an extreme susceptibility of the nervous system, with defective will-power and imperfect self-control; irregular or depraved appetite; flatulent dyspepsia; constipation; interrupted, sighing respiration; rapid action of the heart; varied hyperæsthesias, such as tenderness in the epigastrium or in the course of the spinal column or over the ovary; that peculiar pain in the left side which distresses so many hysterical and anæmic women; and anæsthesia often confined to a circumscribed area, to a single member or to one side of the body, and often profound. Besides these, we encounter manifold local hysterical ailments, such as hysterical paralysis, hysterical aphonia, hysterical tremor, hysterical anorexia, hysterical peritonitis, hysterical affections of joints, hysterical pain in the forehead, hysterical hæmoptysis, hysterical barking cough, hysterical sweating, hysterical suppression as well as hysterical retention of urine. Hysterical laughter has been found to occur on a large scale as a form of epidemic convulsion.[2] There may be hysterical deafness, or hysterical amaurosis, or retinal hyperæsthesia, or crossed amblyopia. J. K. Mitchell and de Schweinitz[3] consider disturbance of color-sense

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[1] Lectures on Diseases of the Nervous System. See also Richer, Études cliniques sur Hystéro-Épilepsie, Paris, 1881.

[2] D. W. Yandell, Brain, Oct. 1881.

[3] Journal of Mental and Nervous Diseases, vol. xix., No. 1, p. 1.

common in hysteria. They have found reversal in the normal sequence of colors to be usually present in cases attended with anæsthesia. Muscular atrophy has been observed as a manifestation of hysteria;[1] a low-grade optic neuritis has led to the supposition of cerebral tumor;[2] and a case has been recorded closely simulating syringomyelia.[3] In hysterical insanity a suicidal tendency is often noticed.

Hysteria is met with in the male, especially after railway accidents. Hysterical paralysis may also happen in either sex, in the shape of hemiplegia, of monoplegia, or of paraplegia, and may be of extremely long duration.[4] As regards hysterical hemiplegia, it is remarkable that it does not affect the face; yet there may be an hysterical facial paralysis.[5] Hysterical headache, Charcot tells us, like syphilitic, increases at night, and is similar to the tremor from metallic poisons. Hysterical tremor is most common in the hands and arms. Hysterical contractures may occur in both arm and leg; complete anæsthesia causes them to disappear temporarily. The reflexes in hysteria may be much deranged. As Goodell[6] well says, strange and misleading reflexes come from the loss of brain control over the insubordinate lower nerve-centres. In *toxic hysteria*, such as we observe after chronic lead or mercurial poisoning, tremor, anæsthesia, palsies, and anorexia or hysterical vomiting are often observed,—much oftener than hysterical paroxysms.

Fever is not a symptom of hysteria. Yet occasionally we meet with cases that it is difficult to explain in any other way, and hysterical disturbance of the heat-centres with extraordinarily high temperatures certainly happen. We may also have fever in hysterical local diseases, as in hysterical meningitis, in hysterical peritonitis.

The distinction between these hysterical pseudo-maladies and the diseases they simulate is far from being an easy task. We have to take into account the patient's age and sex; whether or not she has suffered from paroxysms of hysteria; how the pain is influenced by pressure; the great tendency to exaggeration and deception; and the signs of functional disorder of the apparently affected part. We may thus avoid mistaking a phantom for a true disease. Yet there is another and opposite source of error quite as strenuously to be

[1] Hirst, Deutsche medicinische Wochenschrift, 1894, No. 21, p. 459.

[2] Mills, The Nervous System and its Diseases, 1898, p. 527.

[3] Wichmann, Berliner klinische Wochenschrift, 1895, No. 12, p. 252.

[4] See cases reported by Morton Prince, of twenty-nine, twenty-eight, and twenty-nine years' duration, Amer. Journ. Med. Sci., July, 1892.

[5] Babinski, Société Médicale des Hôpitaux de Paris, 1892.

[6] Medical News, Jan 6, 1894.

guarded against. The complaint may be really an organic one, occurring in an hysterical patient, and concealed, or exaggerated and complicated, by the symptoms of hysteria. In all such doubtful cases we must accord great weight to the extent of functional and constitutional disturbance accompanying the local morbid state. Then, too, hysterical symptoms may be prominent in certain brain and cord affections. I have repeatedly noticed them in cases of cerebral embolism; and Brown-Séquard and Seguin[1] have shown their frequent occurrence in lesions of the right hemisphere. In hysterical attacks connected with a cerebral neoplasm, the urea and the phosphates in the urine, Gilles de la Tourette[2] shows, are diminished, while in epileptic seizures connected with brain tumors they are increased. Hysteria may also complicate myelitis and lateral sclerosis.

Hysteria is sometimes feigned,—feigned to elicit sympathy, or to procure compliance with wishes or caprices. Nor is the simulation of the disorder an outgrowth of our civilization. The epigrams of Martial prove how common the feigning of hysteria was among the Roman women.

Tetanus.—A very fatal disease marked by persistent rigid contraction of the voluntary muscles, particularly of those of the jaw, with violent brief exacerbations.

This distressing malady, as we see it, is generally *traumatic*, following a wound, or an injury; for *idiopathic* tetanus is very seldom met with in temperate climates. But in hot countries, or in those in which sudden alternations of temperature are common, it is not a rare disease, and is indeed frequent among new-born children. The malady is also seen in the puerperal state.

The symptoms of tetanus depend upon the action of a poison generated by a special micro-organism, the bacillus tetani. This is a long, slender bacillus, found commonly in the superficial layers of earth, and growing best in the absence of air and light. It is usually met with in the local lesion. In the so-called idiopathic cases the channel of microbic infection eludes detection.

The muscles ordinarily first affected are those of the jaw and neck; there is a stiffness about them which the patient is apt to attribute to having caught cold. Sometimes, however, the disorder exhibits itself primarily in the external respiratory muscles. When the malady is fully developed, most of the muscles are stiff and hard, the jaw cannot be opened,—whence the common name of lock-jaw,—and there

[1] Archives of Electrology and Neurology, May, 1875.

[2] Quoted, Lancet, May, 1893, p. 1083.

is much difficulty in speaking and in swallowing. The face is distorted, presenting the "risus sardonicus." With these symptoms we usually find rigidity of the muscles of the abdomen and of the limbs, and a distressing pain at the pit of the stomach, dependent upon spasm of the diaphragm. Besides the permanent contraction of the voluntary fibres, exacerbations of spasm take place, during which the muscles become very hard. These paroxysms are accompanied by intense pain, and recur with increased severity and frequency as the disease advances to a fatal termination. When at their height, the body becomes curved, the patient merely resting upon his head and heels. This is *opisthotonos;* while the setting of the jaw, especially when its muscles alone are affected, is called *trismus*. The trunk may be bent forward,—*emprosthotonos;* or to one side,—*pleurothotonos;* or the trunk and neck are rigidly extended in a straight line,—*orthotonos*. The spasm relaxes during natural sleep or induced narcosis. At the height of the attack the body is covered with copious perspiration.

Notwithstanding the striking muscular disorder and the exhausting pain, there is little constitutional disturbance; the pulse may be quickened, but it preserves its volume until the last stage is reached; and there is no fever, certainly not in the earlier stages, nor is the intellect affected. Yet the temperature shows extraordinary variations. The thermometer may mark an increase of several degrees in the evening,[1] and towards the end indicate a heat of 110° F., even continuing to rise after death.

When tetanus results from an injury to the head, and more particularly in the distribution of the fifth nerve, there is often, in addition to the initial trismus, paralysis of the face on the same side as the injury, and spasm on the opposite side.

Tetanus runs an acute or a chronic course. Some cases last three weeks, and when of such long duration are apt to recover. But generally the malady terminates fatally before the eighth day.

Few complaints are likely to be confounded with tetanus; yet these few resemble it in many respects closely. For instance, one of the freaks of hysteria is to take the appearance of tetanus; and tonic spasms dependent upon an affection of the spinal cord or medulla oblongata, strychnine poisoning, or hydrophobia, may accurately simulate its symptoms.

Hysterical tetanus is distinguished from the real disease by being preceded by, or attended with, fits of hysteria; by the age and sex of the patient; by the absence of pain; by the occasional occurrence of

[1] Ogle, Clinical Society's Transactions, 1872.

clonic instead of tonic spams; and by the intermission every now and then of all muscular rigidity. Moreover, the influence of the mind upon the seeming tetanus is very striking. If within hearing of the patient the employment of cold to the spine, or of the cautery, be threatened, or, better still, if the latter instrument be actually made ready for use before her, an extraordinary subsidence of all stiffening and starting of the limbs takes place. Hysterical trismus is more common than extended hysterical tetanoid spasm, but, besides the symptoms of hysteria just mentioned, the absence of rigidity in the neck is very significant.

Tetanic spasms *symptomatic* of an affection of the spinal cord are separated from tetanus by the different history; by no violent exacerbations being brought on, as they are in tetanus, by slight movements, or by an attempt at speaking, or by any reflex irritation; by the absence of marked remissions; by the rigidity being almost always limited to the extremities,—except in the case of meningeal apoplexy in the cervical region, in which the tonic contraction in the upper extremity is associated with stiffness of the neck; by its association with altered sensibility; and by the setting in of palsy before the malady terminates.

In the tetanic spasms which may occur in scarlet fever, in typhus, in smallpox, or in pyæmia, and which are the result of an irritation of the cord produced by the poisoned blood, the rigidity runs so uncertain a course, appears so quickly, disappears so suddenly, perhaps not to return, or only to reappear after a considerable interval, that there is little likelihood of confounding the muscular disorder with tetanus. Tetanus differs from *meningitis* in the absence of pyrexia, of headache, and of vomiting; in the early presence of trismus, which, indeed, in meningitis may be wholly wanting; and in the fact that the spasms in this are generally only induced on attempted movement, whereas in tetanus the reflex irritability is so great that they are induced by the slightest touch. In *cerebro-spinal fever* the resemblance is much closer; yet the whole history of the disorder, the marked headache and mental symptoms, the fever, and the progress of the case, are such as to prevent error. With *muscular rheumatism* tetanus can only be confounded at its onset; but the muscles of the jaw are not rigid in rheumatism.

Another form of symptomatic rigidity requires it to be distinguished from tetanus,—a local rigidity, owing to irritation of the nerve supplying the stiffened muscles; as, for instance, a spasm from irritation of the peripheral or the central tract of the motor portion of the fifth, the so-called *masticatory spasm* of the face. The ailment may be of reflex origin, the exciting cause being a decayed tooth, a wound, or

exposure to cold; or it may exist in connection with apoplexy, or with *an inflammation of the brain*. Its main marks of distinction from the *trismus* of *tetanus* are, that it is purely local, is often of long continuance, is not painful, has no paroxysms of aggravation, is not combined with impaired deglutition, and is not dangerous.[1] Similar spasm has also been observed as a result of irritative lesions of the pons, such as a new growth, or vascular disease, affecting the motor nucleus of the fifth nerve.

Tetany is characterized by tonic contractions, more especially of the legs and arms, which may be intermittent, remittent, or continuous; the toes are apt to be flexed towards the soles; the hands become fixed; the spasm, dissimilar to what happens in tetanus, begins in the extremities. The jaws and the respiratory muscles are, unlike what we find in tetanus, not affected, or the jaws become so only towards the end in severe cases.

The spasms are painful; they may occur several times in a day, or there may be weeks between them. They also can be produced, as Trousseau discovered, by pressure on the chief arteries and nerves of the affected limb. They are usually preceded by tingling or burning; in the intervals between them the muscles are readily excited to contraction and there is increased electrical excitability; the temperature, as a rule, remains normal throughout, although in severe paroxysms it may be elevated, and there may be copious perspiration. The irritability of both sensory and motor nerves is increased; and the remarkable irritability is shown by *Chvostek's symptom*,—a slight tap in the course of the nerve, as of the facial, will throw the muscles to which it goes into strong contractions. The contractions in tetany are bilateral, which distinguishes them from hysterical contractures. Tetany differs from *carpopedal spasm*, observed in rickets or in severe gastro-intestinal catarrh, by the spasms of this being much more transient. They are also apt to be much more marked in the fingers than in the toes. But the distinction is chiefly one of degree, and many regard carpopedal spasm as only a light form of tetany. This malady happens chiefly in suckling children, as a sequel of exhausting diarrhœa, or exposure to cold, and in nursing or in pregnant women: it has also been observed in connection with dilatation of the stomach and other lesions of the internal glands. It has been described as occurring in epidemic form, and the symptoms are like those of

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[1] Spitzka, in the second volume of his Boston Series, gives the particulars of a case which illustrates many of the difficulties in diagnosis in which the affection may give rise.

ergot poisoning.[1] The disease is not a common one in this country. Crozer Griffith[2] has analyzed seventy-two cases reported in America.

The symptoms of *strychnine poisoning* are almost identical with those of tetanus; yet there are some characteristic differences. The spasms from strychnine do not supervene upon exposure to cold, or upon a wound, but follow within about two hours or less the taking of some solid or liquid. They come on suddenly, with violence, with epigastric pain and early reflex excitability. The tetanoid convulsions affect simultaneously nearly all the voluntary muscles of the body, but with greatest intensity those of the trunk and spine, producing very early—within a few minutes, commonly—a marked opisthotonos, which in tetanus does not appear, if it appear at all, for many hours or days after the seizure. On the other hand, the stiffness of the jaws, which is among the very earliest signs of tetanus, is not at first perceived in strychnine poisoning, and, if it occur, occurs only imperfectly. Further we do not see the frightful tetanic face, with its knit brow and horrid grin; we do not observe intermissions in the convulsions, or difficulty in swallowing; and in from ten minutes to two hours after the commencement of the attack the patient dies or recovers.

Finally, let us contrast tetanus with *hydrophobia*. Both showing the reflex functions of the spinal cord to be in an exalted condition; both being spasmodic affections lasting ordinarily but a few days; both taking place, the popular opinion to the contrary notwithstanding, at all periods of the year; both presenting violent paroxysms of convulsions, which are often excited by the slightest touch or jar to the body; both frequently occasioning torturing pain near the pit of the stomach; both ensuing commonly upon an injury; both usually augmenting in intensity from hour to hour,—these ghastly maladies are yet dissimilar. The one results from infection with a specific bacillus often present in earth; the other from infection with the virus of a rabid animal, most commonly the dog or the wolf. The one has a short, the other a long period of incubation. In the one, deglutition may be difficult; in the other, it is next to impossible, all attempts at swallowing, especially of fluids, exciting the most distressing spasmodic dysphagia. In the one, early rigidity of the muscles of the jaw happens; in the other, there is no such rigidity. In the one, the breathing may or may not be interfered with; in the other, the spasms of respiration are almost as marked a feature as

[1] Stated in the German translation of this book.

[2] Trans. of the Assoc. of Amer. Phys., vol. ix., 1894.

the spasms of deglutition. Then the irritability of temper in hydrophobia; the fierce manner of the patient; his rabid, perhaps maniacal paroxysms; the constant thirst; the accumulation of stringy mucus about the angles of the mouth; the vomiting; the acute sensibility of the surface; the trembling of the muscles; the clonic instead of tonic spasms; the husky voice; the strangling sensation in the throat,—are phenomena too striking to render an error in diagnosis likely. The temperature is, as a rule, elevated, and in direct proportion to the intensity of the other symptoms. Towards the close it may reach a high degree, and it sometimes continues to rise after death. Some of the points here referred to serve also to distinguish hydrophobia from acute mania, and from hysteria. For, as in tetanus, we find this erratic complaint simulating the terrible disease. In truth, it is the opinion of some, of Dulles[1] especially, that the great majority of cases of supposed hydrophobia are of this character.

Functional Spasms.—There are spasms that take place in various parts of the body, sometimes clonic spasms, sometimes tonic spasms, which occur without apparent cause, and are more or less continuous or persistent. In time they may lead to contractures and deformity, or they may pass away. They may be of hysterical origin; but these are not now under discussion, rather the spasms that take place in one or both legs, sometimes in the arms, occasionally in the muscles of the face, which occur in those who are not hysterical subjects, and are not traceable to any lesions. Pressing on particular points may at once excite them; on the other hand, there are "pressure-points" which when acted on will cause the convulsive movements to be arrested. The trophic disturbance that attends them is usually very slight. Tonic contractions are apt to alternate with clonic spasms, or there may be only complete tonic spasm during attempts at moving certain muscles. At times spasms of the internal muscles, as those of deglutition or respiration, may coexist; or the spasms may be limited to these muscles. The disorder is sometimes hereditary.

There is a curious form of spasm, a tonic contraction of the muscles, which impedes locomotion. It shows itself when the muscles are first put into action after a period of rest, or after an unexpected irritation, as striking the toes against a stone in walking, and is augmented by nervous dread about it. Happening, as it generally does, in the lower extremities, it leads there to muscular increase. This

[1] Transactions of the College of Physicians of Philadelphia, 3d Ser., vol. xvi., 1894.

Thomsen's disease, or congenital or *transient myotone*, has been known to originate in sudden fright.[1] It begins commonly at an early age, and is hereditary; it is persistent, although no organic cause for it has been detected. In one fatal case post-mortem examination disclosed the existence of hyperplasia of the muscular tissue, without appreciable lesion of the central or peripheral nervous apparatus.[2] The difficulty is most marked in the morning, on first rising, attempts at movement causing the muscles to become rigid and the joints fixed: yet if exertion be persevered in, the spasm becomes less and less, and continued walking is possible until after another period of rest. The spasm is aggravated by attention and emotion, and very rarely affects the muscles of the face. Electrical and mechanical irritability are heightened. Sensibility and reflex excitability are unaffected.

The chief difference between Thomsen's disease and *paramyotone*,[3] which is also a family affection, is, that in the latter spasm is not started by voluntary movements and is more permanent; the marked spasms may last several hours. They are excited by cold and allayed by warmth. Paramyotone may be associated with ataxic symptoms.

A family type of congenital myotone is described by Eulenburg,[4] and intermittent congenital myotone by Martin and Hausemann,[5] in both of which exposure to cold was followed by tonic spasm of various muscles. The irritability of the affected muscles was diminished. It is not impossible that future investigation may disclose some relationship between the spasmodic conditions here described and some of the forms of functional palsy previously detailed.

Hiccough.—As a form of local spasm may be here mentioned the curious phenomenon called hiccough, an intermittent, sudden contraction of the diaphragm. It is a matter of doubt whether this is connected with irritation of the respiratory centre, or is a spasm from irritation of the phrenic nerve, reflected, it may be, from the pneumogastric, or direct. Its symptoms are a spasmodic contraction of the diaphragm, followed by a sudden closure of the glottis with a short, cough-like noise. It may occur in brief paroxysms of varying duration, or go on by day and night, and result in wearing out the

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[1] Case of Schönfeld, Berliner klinische Wochenschrift, July, 1883.

[2] Déjerine and Sottas, Compt.-rend. hebdom. des séances de la Soc. de Biol., 1893, No. 23, p. 669.

[3] Gowers, Diseases of the Nerves and Spinal Cord, vol. i., 1899.

[4] Neurologisches Centralblatt, 1886, No. 12; Jahresber. über die Leist. u. Fortschr. i. d. ges. Med., xxi. 2, p. 164.

[5] Arch. f. path. Anat. u. Physiol. u. f. klin. Med., cxvii. 7, p. 587; Jahresber. über die Leist. u. Fortschr. i. d. ges. Med., xxiv. 2, p. 76.

strength of the patient. It is met with in various affections, both of the nervous system and of the œsophagus, stomach, and intestines. In some instances it is clearly of rheumatic, or gouty, or uræmic, or other toxæmic origin. When traceable to the nervous system, it may be centric, due to the pressure of localized inflammatory exudation, or of a new growth, or be the result of reflected irritation. When met with in diseases of the stomach, the irritation is peripheral and clearly reflected. In persons with atonic and flatulent dyspepsia or catarrhal conditions of the stomach, hiccough is not an uncommon symptom. Hiccough is also seen in diaphragmatic pleurisy, in dysentery, in appendicitis, in peritonitis, and in disease of the heart. Irrespective of the causes that are distinctly centric, or are peripheral and reflected through the pneumogastric nerve, cases of hiccough occur that cannot be traced to any obvious cause, and in which it appears as a pure neurosis. These are apt to be among the most obstinate ones; many of them occur in hysterical subjects.

Diseases of Ill-Regulated or Deficient Nerve-Force.

The diseases which principally belong here are hysteria and neurasthenia; in both there is also marked psychic perversion. Hysteria has already been described in its most striking form,—the convulsive. This brings it into the group marked by convulsions or spasms, with which it is most conveniently considered.

Neurasthenia.—The weakness of the nervous system shows itself as a general state of nervous exhaustion, or as the nervous weakness of special parts, such as cerebral neurasthenia, spinal neurasthenia, sexual neurasthenia, gastro-intestinal neurasthenia, cardiac neurasthenia, and vasomotor neurasthenia. There are no strict differences between these forms, and, even in these local manifestations, the evidences of a general neurasthenic state may be found. Neurasthenia may be from inborn nervous weakness; or acquired from strain of life, anxiety, worry, loss of sleep, eye-strain, from effort made too soon after exhausting disease, from prolonged mental labors undertaken by those who lead in-door lives; or traumatic, as seen especially after railway accidents.

The symptoms of the general neurasthenic state are manifold. There is generally weakness, which becomes painfully manifest on any sustained bodily or mental exertion, some loss of weight, and a depressed or despondent look. Anæmia and, in women, hysterical manifestations are of frequent occurrence, and low spirits are general, though cheerfulness is sometimes met with. As a rule, there is insomnia, as well as a sense of weight and pressure in the head, pain at the back of the

neck, and inability to fix the attention on any subject for long, certainly to do so without fatigue. All business affairs, even the smallest, become a source of worry, of annoyance, of self-reproach. The temper is irritable, and the patient is constantly talking of himself, his symptoms, his sufferings. The appetite may remain good, but it is often impaired, and there is a condition of nervous dyspepsia with lithæmic urine. The eye is sensitive, and aching in the eyeball after reading or flashes of light are complained of; there is, indeed, a very familiar neurasthenic asthenopia. Inequality of the pupils and their dilatation and drooping of one eyelid are usual. Both the deep and superficial reflexes are increased, and hyperæsthesia, especially increased sensitiveness to pain, is common. Then there are pain or aching in the back and spine, and various neuralgic pains and dizziness. In some instances the psychical phenomena preponderate. There is intense dread of one object or the other, of people, of things, of disease, of insanity, curious and uncontrollable thoughts run through the mind, and suicidal tendencies may show themselves. The will-power is greatly impaired; there is an inability to come to any decision. The vasomotor disturbances show themselves by flushes of heat, sweating, and throbbing of the abdominal aorta, as well as general arterial throbbing and imperfect capillary circulation, with coldness of hands and feet and numbness.

Accordingly as one or the other group of symptoms preponderates, we have different local types established, and among these the cerebral and the spinal may be very confusing, and readily lead to the supposition of organic disease.

The *cerebral* I described years ago in the first edition of this work (1864) as "exhaustion of brain power," and pointed out how it differs from the phenomena generally attributed to softening of the brain. It is encountered among overworked professional men or those engaged in laborious literary undertakings. It sometimes comes on suddenly, with signs like those of collapse; more generally it is slower in development. Its manifestations are a slight deterioration of memory, and an inability to read or write, save for a very short period, although the power of thought and of judgment is in no way perverted. Nor is the power of attention more than enfeebled: the sick man is fully capable of giving heed to any subject, but he soon tires of it, and is obliged from very fatigue to desist. He passes sleepless nights, is subject to ringing in the ears, cannot bear much exercise, is troubled with irregular action of the heart, with a frequent desire to urinate, and with neuralgic pains in the face or a feeling of soreness in the head; but he does not generally lose flesh, and his digestion is unimpaired.

Many remain in this condition for months, and then slowly regain their health. What the precise disturbance of the brain consists in is uncertain: it is possible that the nutrition of the organ has been interfered with from overuse and worry. The phenomena of this *cerebral neurasthenia*, as it is now customary to call the disorder, differ from those of softening by the absence of, or at least by the far less permanent and marked, headache, by the comparatively unimpaired intelligence, and by the non-occurrence of spasms or of paralysis, and of the causes that generally produce softening.

Cerebral neurasthenia may be mistaken for the earlier stages of general paresis. But though they have signs of nervous weakness and exhaustion in common, and even some of the psychic manifestations, yet the slowness of speech, the tremor of the tongue, the condition of the pupil, generally myotic and with impaired reflex, and the change in character denote the paretic affection, in which, moreover, there is often a history of syphilis or of alcoholism. In the more advanced stages of the malady the impaired gait, the almost unintelligible speech, and the delusions leave no doubt that we are not dealing with neurasthenia.

Spinal neurasthenia manifests itself by pain and tenderness of the spine, intercostal neuralgia, aching pain in the legs, numbness and tingling in the extremities, some defect of co-ordination, as shown in the gait and in writing, and these symptoms may simulate beginning locomotor ataxia. But there is no Argyll-Robertson pupil, there are no lightning-like pains, and no sensory disorders, for the sensation is only subjectively disturbed, and the reflexes are either normal or increased.

There may be much difficulty in distinguishing these cases of spinal neurasthenia from those of so-called *nutritional disease of the spinal cord*, which Gowers has specially described. The symptoms, indeed, are the same, except that aching in the back and legs is more pronounced; that aching in the legs at night is complained of; that there is always increased knee-jerk; that even the shortest walks produce at once a sense of fatigue in the legs; that these show some falling off in nutrition; and that there is a history of a fall, or of an acute illness, such as typhoid fever or acute rheumatism, or of sexual excesses. Then the general neurasthenic symptoms are absent or are not marked.

In *sexual neurasthenia* there is great irritability of the sexual organs, prostatorrhœa and spermatorrhœa are complained of, and their importance immensely exaggerated; there is pain in the testicles, also generally greatly exaggerated, and constant dread of im-

potence. In women, derangement of the menstrual function is not uncommon.

The diagnosis of neurasthenia is frequently difficult, as we have to depend so much upon the statements of the patient. Moreover, it is a diagnosis often incorrectly made by the physician, and too readily acquiesced in by the patient, who is not loath to believe himself the victim of "nervous prostration." This is, indeed, one of the greatest of difficulties; the lazy, the irresolute, the self-indulgent have a name under which they dignify their failings, or shelter their shortcomings. Then ill health associated with the beginnings of organic disease in various organs is very apt to be pronounced neurasthenia. No diagnosis of this ought ever to be made until after the closest search for a structural affection, and especially for lesions in the nervous system, kidneys, stomach, and blood; a large number of cases of so-called neurasthenia turn out, indeed, to be a disease of one of these parts. Neurasthenia is most apt to be confounded with hysteria and with hypochondriasis, and what makes the diagnosis at times very perplexing is that there may be an association of the morbid states. In hypochondriasis, almost exclusively a disease of males, there are actual delusions concerning the physical state, which may, however, be very good; not so in neurasthenia, though there is often great dread of disease. The paroxysms of hysteria, its peculiar mental characteristics of exaggeration and deception, the emotional disturbances, the crises, the alterations in vision, the contractures, the anæsthesias, the hysterical palsies, and the great range of hysterical symptoms distinguish it.

Diseases characterized by Gradual Impairment of the Mental Faculties with Paralysis.

Chronic Softening.—Softening of the brain may be caused by nutritive changes consequent upon a diseased state of the cerebral vessels, or by an inflammatory disease spreading from the meninges to the brain, or taking place around new formations and old lesions. It may also follow cerebral hemorrhage. But its chief cause is occlusion of the cerebral arteries from embolism. In rarer instances the plugging is due to a thrombosis. The middle cerebral arteries are the most common site of the emboli, and degeneration and softening occur in the territories supplied by the obstructed vessels. Whatever the cause of the softening, the symptoms are much the same. They are briefly these: gradual impairment of intelligence; weakening of memory; headache; vertigo; muscular debility; cutaneous hyperæsthesia or anæsthesia; formication and numbness; and slight

or partial palsies, particularly of the muscles of one side of the mouth, or of one eyelid. Then there is not unfrequently defective articulation, with great irritability of temper, nausea and vomiting, extreme sensitiveness to sounds, and painful feelings in various parts of the body. As the local mischief advances, the paralysis becomes more decided, assuming generally the hemiplegic form; and spasms, either tonic or clonic, or epileptic convulsions, occur.

In the diagnosis of softening the most important point is the recognition of the state that has led to it, the meningo-encephalitis, the apoplexy, the diseased blood-vessels, or, above all, the embolism which has started the process which, in place of an acute course, is pursuing that of a slower degeneration. The older descriptions of softening are very fallacious. Many cases of cerebral neurasthenia, many of general paresis, were covered by this term, and the secondary results of morbid processes in the brain were looked upon as the primary disease.

We shall next inquire how such cerebral maladies as congestion, anæmia, abscess, and atrophy may be distinguished from softening.

Congestion is discriminated by its being very rarely a persistent state. It may be active or passive,—resulting on the one hand from an increased supply of blood, and on the other hand from interference with the venous return. An acute attack produces the symptoms of apoplexy; a more lasting congestion is recognized by tracing the cause which has led to the fulness of the vessels,—such as a disease of the heart or of the abdominal viscera,—and by noting that, although the patient suffers from dull headache, from disturbed sleep, from jerking of the muscles, from pulsation of the carotids, from vertigo, these signs are far from constant, and come and go for a long time without any material disturbance of the functions of the brain being perceptible. The finding of optic neuritis or choked disk, or the presence of paralysis, would determine against congestion.

Cerebral anæmia, occurring suddenly, produces unconsciousness, or dizziness or stupor; or, if general, convulsions. When more gradually induced, it manifests itself by drowsiness, sighing respiration, distressing headache, often referred to the vertex; by a pale face and uninjected eye with large pupil; by derangement of the special senses; by the vertigo and the other symptoms of cerebral disorder being relieved in the recumbent position; and by a feeble pulse and cool forehead. Then, in tracing its history we are apt to find that it occurs in those who have been exhausted by debilitating diseases, or by repeated hemorrhages, or by albuminuria. The chief distinction from softening lies in the history of the case; the aspect

of the patient, and the absence of palsies, or their passing nature, must be taken into account. But we must not forget that anæmia is also the first stage of softening due to vascular occlusion.

Abscess of the brain arises under the same conditions as cerebritis; but pyogenic micro-organisms play a very important part in the morbid process. The most constant clinical association is with disease of the ear; suppurating processes in other parts of the body, such as abscesses in the lungs or fetid bronchitis, are also not infrequent causes. The symptoms are referable in part to the inflammatory process, in part to the presence of the purulent accumulation. The acute cases get progressively worse; in cases which pursue a chronic course an initial inflammatory stage of brief duration is succeeded by a latent period, sometimes of considerable length, and this in turn by a terminal stage, ending rapidly in death. Among the early symptoms are headache and vomiting, in association with febrile disturbance often attended with chills. Involvement of the cortex or subjacent white matter may cause local spasm; extensive disease, general convulsions. Paralysis and delirium may be also present. The acute period lasts from a few days to several weeks. In the latent stage, which may continue from a month to some years, decided manifestations may be wanting. Often there is headache; occasionally there are convulsions; at times slight mental disturbance exists. Elevation of temperature, and recurrent rigors followed by sweats, also happen in abscess. Optic neuritis is as often absent as present. Constant headache and vomiting are among the most prominent symptoms. Though hemiplegia is met with not unusually, it is generally slight. The terminal stage which marks the rupture of the abscess may set in abruptly or gradually, with increase in the headache and mental symptoms, with vertigo, vomiting, derangement of consciousness, convulsions, and paralysis.

Few cases of abscess of the brain, as Lebert[1] has shown, last longer than eight weeks. Abscess of the brain may be latent, and the sudden rupture of the abscess may give rise to symptoms undistinguishable from those of hemorrhage, undistinguishable unless we can infer an abscess from a disease of the bones of the head, or from some points in the history of the case.

Atrophy of the brain is especially observed in old age, and, when marked, may be the cause of the general decay of cerebral functions noticed at this period of life. It is very generally connected with

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[1] Archiv für Path. Anat., Bd. x. See also Gull's paper in Guy's Hospital Reports, 3d Series, vol. iii.

diffused sclerosis. As a rule, it occasions no distinctive symptoms; it has been specially observed in idiots. The brain is sometimes undersized from defective development. The diminution may be general or unilateral, or even circumscribed. Partial atrophy is a common result of meningeal hemorrhage during birth. In some instances it follows meningitis early in life. Similar processes may also take place during intrauterine existence. These varying conditions give rise to diverse symptoms, among the most common of which are mental defect, hemiplegia, convulsions and mobile spasm.

The differences between softening and *cerebral neurasthenia* have been already considered, and those between it and tumor will presently engage our attention.

Tumor.—Tumors of the brain give rise to a great diversity of signs, according to their locality, their size, and their nature. Let us first examine the symptoms by which we may infer their existence.

The presence of a tumor in the brain is rendered probable if, in addition to vertigo, to vomiting or to a disposition to vomit, or to headache, violent, but paroxysmal and neuralgic in its character, we find impairment or loss of vision, or indeed of any special sense, and epileptiform convulsions not followed by any greater deterioration of health than previously existed; if with these signs of cerebral irritation the intellect is not at first markedly disordered, nor the articulation affected; and if paralyses do not show themselves until a long time after the headache, and are even then limited to the muscles of the eyeball or of the face, or to the muscles of the extremities of one side of the body. As a further sign of cerebral tumor we may class optic neuritis or choked disk. It is a curious fact to be borne in mind that cerebral tumors occur in males more than twice as frequently as in females. It may also be noted that the larger number of cases are in the young or in those in the prime of life; the aged are remarkably exempt. The commonest forms of tumor are tuberculous, gliomatous, sarcomatous, and syphilitic. Less common are carcinomata and parasitic tumors. Before the evidence of a tumor is considered conclusive we must exclude other chronic cerebral maladies, especially softening, abscesses, and chronic meningitis.

We separate chronic *softening* by noticing that the headache caused by a tumor is much more constant and violent, having paroxysmal exacerbations; that the intelligence remains for a long time intact, save, perhaps, in a weakening of the memory; that optic neuritis is a usual accompaniment; that motor and sensory disturbances are less frequent and prominent, but convulsions far more so. Further, cerebral tumor is more common in early life, chronic softening in late life.

Disease of the heart or of the blood-vessels, or Bright's disease, or, especially, the history of an embolic seizure, points to the latter state. Remissions, or intervals of apparent improvement, occur in both morbid conditions; but they are more perfect and of longer duration in tumor than in softening.

The differential diagnosis between tumor and *abscess* is more difficult. We may conclude the latter to exist, if the cephalalgia be sudden in its development, and uniform and general, instead of neuralgic and limited. Then epileptic convulsions, drowsiness, paralysis and coma succeed one another much more rapidly and, except convulsions, are present much more constantly in abscess than in tumor,—indeed, epileptic fits are about as often absent as present.[1] Further, optic neuritis and localizing symptoms are more common in tumor than in abscess, and this shows especially in the palsies of cranial nerves. If, moreover, we obtain the history of injury to the skull, or find a discharge from the ear, or pain upon pressure over the mastoid process, or a chronic disease about the head, or protracted suppuration in any part of the body, we may safely infer that an abscess, not a tumor, is the cause of the evident cerebral mischief. Abscess, like tumor, chiefly affects males.

Chronic meningitis, an affection sometimes complicating tumor, is discriminated by laying stress on its etiological relations,—such as blows upon the head, diseases of the cranial bones, syphilis, rheumatism, alcoholism, chronic tuberculosis,—and by observing its frequent though irregular accessions of fever, the great irritability of temper, the dulness of intellect, the loss of memory, and the nocturnal delirium. The pain, too, is duller and more diffused than in tumor, and there is more vertigo. The localizing symptoms are not so definite and fixed, nor the convulsions as distinctly epileptiform in type. Yet convulsive movements of some muscles are common, and may be even followed by incomplete paralysis. Meningitis may be excluded if optic neuritis or any marked alteration of the disks be found early in the case. Indeed, optic neuritis is absent or is very slight in chronic meningitis. Yet the diagnosis is often very difficult, especially between tumor and syphilitic or protracted tubercular meningitis.

Thrombosis of the sinuses of the brain may occasion partial palsies, with symptoms of cerebral pressure, like those of tumor, and cannot be distinguished except in the instances in which we find distention of the collateral circulation shown in the fulness of the veins of the

[1] Thus, they occurred in only thirty-eight cases of abscess of the brain out of seventy-three collected by Gull and Sutton (Reynolds's System of Medicine).

nose, temple, and forehead, and injection and œdema of the forehead and eyelids. Convulsions, further, are very rarely among the symptoms; and generally these are more similar to the manifestations of meningitis than of tumor; coma is not uncommon. When primary, the condition is usually a result of enfeeblement of the circulation and altered blood state in exhausting or wasting diseases, especially those of infancy and old age. In children with marasmus, or in adults with caries of the skull, or purulent ear disease, marked cerebral phenomena may lead to the correct inference of thrombosis. Secondary thrombosis is most often met with as an infective process from adjacent disease, especially chronic suppurative disease of the ear, and there is local œdema and tenderness over the mastoid and internal jugular. Portions of the disintegrating thrombus may be carried into different parts of the body, and embolic phenomena appear. In the marasmic cases the symptoms are often those of the hydrocephaloid disease of Marshall Hall, with which it may be associated; hemorrhage into the cortex of the brain is common.

The precise *seat* of the tumor is difficult to determine. An affection of the special senses or of cranial nerves points to disease near to, or at, the base of the brain; and the probability of this view is much strengthened if there be paralysis of the face on the side opposite to that of the extremities, and if vigorous inspiration, during which the brain falls and presses the morbid mass against the walls of the base of the skull, cause or increase pain; whereas, so says Romberg, in tumors on the upper surface, forced expiration produces a like result. In cases of tumor of the *pons* or the *crus*, particularly when tubercular, incoördination of the arm similar to the jerky movement of disseminated sclerosis is met with; but it is unilateral, not bilateral as in sclerosis. In tumors of the *cerebellum* we have headache, severe vomiting, nystagmus, staggering gait, spasms, and rigidity; the knee-jerk may be absent or increased; there may be no marked alteration of the optic disks, or, as de Schweinitz has pointed out, the appearances may be those usually regarded as indicative of albuminuric retinitis. Tumors in or near the *cortex* of the brain give rise to localized convulsions on the opposite side of the body. In tumors of the frontal lobes there are marked psychical symptoms; and ataxia, such as we observe in cerebellar disease, is, Bruns has proved, a very significant symptom. In tumors of the Rolandic region monospasm and unilateral spasm precede or attend the increasing paralysis. In determining the exact position of brain tumors we must make use of the researches on the localization of the cerebral functions. The difficulty of applying this extending knowledge to the diagnosis of

tumors at the bedside is, that they may give rise to circumscribed inflammation around them, or to irritation in even more remote parts, and that the special manifestations of the disorder of the part affected by the tumor are thus blurred or obscured. Then we must also bear in mind that several tumors may be present.

In endeavoring to determine the seat of the tumor it is necessary to distinguish as clearly as possible the difference between the results of generalized pressure or distant effects, and those due to direct and localized influences. It is only the constant abnormal symptom that points out the location of the lesion. Paralyses, pareses, spasms, which change in intensity or affect now one, now another set of muscles or organs, show that the centres are disordered only indirectly and temporarily, and that the true position of the neoplasm is to be sought elsewhere. Another indication is derived from a consideration of the relative intensity of the different symptoms. The less complete a paralysis, or the less energetic the spasm of a certain set of muscles, the less certain is the injury to be localized in their centres, and the reverse. Too much dependence must not be placed on the subjective location of the pain. Diffuse pressure may cause more pain at a point far removed from the growth than its immediate neighborhood. But when spasm or paralysis of a limited set of muscles exists, as in cortical epilepsy, and the pain is located by the patient at a point corresponding to the topographical position of the corresponding centres, the deduction becomes quite certain that the lesion is at this point. When from other indications the inference is probable that the growth is in the cortical substance, the additional symptom of pain makes the diagnosis more sure.

It is manifest that in all tumors of the cortex, or of the white substance immediately beneath, the symptoms will be *unilateral* and include convulsions. When both sides of the body are about equally affected, the tumor must be placed at the base of the brain, unless the growths be multiple and situated in symmetrical parts of the two hemispheres. Where the symptoms are more intense upon one side of the body than upon the other, the weaker symptoms are to be attributed to the distant or indirect effects of pressure. Paralysis, of course, is a symptom of more profound disturbance than spasm or convulsive movement. The last is therefore probably due to an irritative or indirect effect, or to a slowly growing neoplasm. The existence of *papillitis*, *optic neuritis*, or *choked disk*, is in a suspected case of tumor among the most conclusive signs of intracranial neoplasm. But, unfortunately, it gives scarcely an indication either of the nature or of the seat of the new growth. Yet since the papillitis may precede

other symptoms, since also no deterioration of vision may have been noticed by the patient, a careful opthalmoscopic examination should always be made when there is any thought of the existence of tumor.

Can we form an opinion of the *nature* of a tumor of the brain from any of the signs referable to the cerebral malady? We cannot: the character of the pain has been thought to be of great significance; but the testimony to prove that it is so is in the highest degree unsatisfactory. We may sometimes, however, from the history of the case, or from the existence of some of the manifestations of special cachexia, draw a correct inference. In *gliomatous* brain tumors, Virchow has pointed out, there is often the history of a blow. They are usually single, and most common in the cerebral hemispheres, and occur next in frequency in the cerebellar hemisphere; then in the central ganglia, pons, medulla, crus, and corpora quadrigemina. Gliomata are comparatively frequent in children. *Sarcomata* develop in the brain or in the membranes, or from the bones, particularly at the base. They differ from gliomata in being circumscribed and not infiltrating. *Tuberculous growths* are often multiple and most frequent in the cerebellum; they are also found in the pons, central ganglia, crus, medulla, and corpora quadrigemina.

If we find disease of the lungs, or any evidences of scrofula, and the patient be young, we shall probably be right in conjecturing the tumor of the brain to be a mass of *tubercle;* but if the sufferer be advanced in years, and exhibit tumors in various parts of the body, or other signs of a cancerous diathesis, we may with reasonable certainty presume the tumor within the skull to be *cancerous*. *Syphilitic* tumors are mostly cortical, rarely cerebellar, grow rapidly, and are greatly influenced by antisyphilitic treatment. Other kinds of tumors and deposits can scarcely be said to be within the reach of diagnosis. *Cysts* seated in the superficial portions of the brain either occasion no symptoms or give rise to headache, to attacks of vertigo, to vomiting, and to epileptic seizures, but very rarely to palsies. The symptoms mentioned are far more apt to be present when the cysts occupy the lateral ventricles; then epileptic convulsions are rarely absent.

The manifestations of an *aneurism* within the cranium are those of an ordinary tumor, and the affection is not distinguishable except when the symptoms are referable to the presence of a tumor in the course of a cerebral vessel, and we find present a cause of aneurism, such as syphilis or chronic endocarditis with vegetations, or decided indications of disease of the vessels in other parts of the system.[1]

[1] James H. Hutchinson, Pennsylvania Hospital Reports, vol. ii.

A small aneurism may occasion no symptoms; one large enough to exert pressure on adjacent structures may be attended with headache, often pulsating, usually continuous, sometimes paroxysmal; vertigo; mental dulness and irritability; occasionally convulsions; paralysis; bilateral hemianopsia;[1] rarely optic neuritis. Neither the presence nor the absence of a subjective feeling of pulsation and of a murmur, whether in the carotids or the vertebral or the basilar arteries, and audible on auscultation of the skull, has a positive significance; for, notwithstanding the cases of Jonathan Hutchinson[2] and Humble,[3] in which the diagnosis was made during life, the detection of a murmur, as I know from observation, is not a certain sign. A murmur, moreover, is not uncommon in rickets. Even a pulsating tumor protruding through the skull may not be due to an aneurism, but be caused by a glioma, as in the case mentioned by Mills.[4]

Aneurism of the internal carotid artery may cause blindness on the same side, paralysis of the third, and of the ophthalmic division of the fifth, nerve, impairment of the sense of smell, and hemiplegia. Aneurism of the anterior cerebral may occasion many of the same symptoms, although the muscles of the eyeball usually escape. Aneurism of the middle cerebral is usually attended with hemiplegia and convulsions. Aneurism of the basilar artery causes extensive damage, with widespread paralysis, including the cranial nerves; convulsions are rare.

General Paralysis.—This fatal cerebral malady, known also as general paresis and dementia paralytica, is the result of a diffuse interstitial meningo-encephalitis; the spinal cord may become secondarily affected. Clinically, the disorder is marked by impairment of the powers of locomotion; by an inability to articulate distinctly,—a symptom which precedes the deranged locomotion; by the expressionless countenance; and by failure of memory and complete perversion of the mental faculties, amounting, in fact, to insanity.

The palsy is peculiar: indeed, except towards the end, there is, in the usual sense of the term, no palsy in the limbs at all; there is rather a want of control over their co-ordinate action, displaying itself first in the hands by clumsiness of movements and irregular handwriting, and in the gait by uncertainty and a swaying from side to side when the patient attempts to walk. The impairment of the muscular movement gradually extends: tremulousness in the muscles

[1] Case of Mitchell and Dercum, Nervous Diseases by American Authors, 1895.

[2] British Medical Journal, April, 1875.

[3] London Lancet, Oct. 1875.

[4] The Nervous System and its Diseases, 1898.

of expression is noticed; the speech becomes more inarticulate, until scarcely a word can be distinguished; and the patient cannot rise without being assisted. The reflexes are not uniformly affected; the knee-jerk is often exaggerated, but in some cases reflex contraction of the tendo Achillis is wanting. As the disease advances, the cutaneous sensibility is greatly diminished or is lost. The pupils are unequal, generally contracted and sluggish. The mental derangement is often manifested by an exaggerated sense of personal power or importance, and fancies of great wealth; the moral feelings greatly deteriorate; sometimes there are maniacal outbreaks and epileptic attacks, or alternating periods of excitement and depression. Deceptive remissions in the progress of the disease may take place, but the termination is invariably fatal. Death is often preceded by convulsive attacks and by coma, or by painful contractions of the muscles of the trunk or the extremities, or by obstinate diarrhœa, or by pulmonary affections. Pneumonia is especially common.[1]

The early signs of general paralysis of the insane are difficult to recognize. A change in character, in moral sense, in power of mental attention, and in judgment, absent-mindedness, and weariness easily brought on by brain-work or by any physical exertion, are very significant in a middle-aged man, if joined to alteration in handwriting and some impairment in executing delicate muscular movements. With these symptoms there is commonly, as Folsom[2] mentions, loss of flesh.

In more advanced stages there is not much doubt about the malady. It differs from other forms of extensive general paralysis in being far less of a real palsy. It is certainly far less complete than the extensive paralyses which follow lesions of the upper portion of the spinal cord, or which are consequent upon the poison of lead, or of malaria, or of diphtheria. Its association with marked disturbance of the intellect and its psychic symptoms furnish, moreover, a differential test of great value, and not merely with reference to the general palsies just mentioned, but also as regards neurasthenia, the trembling movements of old age, of progressive muscular atrophy, and of chronic alcoholism. In one of its forms, as Westphal points out, there is a strong resemblance to locomotor ataxia in the signs of disturbed co-ordination, sensory impairment and absence of knee-jerk, with incontinence of urine; but the tremor in the muscles of the lips and face and the perverted mental state become of greatest

[1] Crichton Browne, Brain, Oct. 1883.

[2] Transactions of Association of American Physicians, 1889.

significance. In some cases, moreover, changes in the posterior and lateral columns of the cord have been found after death, in addition to those present in the brain. On the other hand, the ataxia and the palsies distinguish the disease from mere senile dementia. Then, too, general paresis is a disease of early manhood and of middle age, and follows syphilis, mental overstrain and anxiety, alcoholism, or sexual excesses.

The defect in the articulation and the attending tremor of the lips, and in some instances the occurrence of apoplectiform seizures, accompanied by considerable elevation of temperature, may cause the disease to be mistaken for *cerebro-spinal sclerosis*. But in this affection, while the embarrassed, scanning speech coexists with great helplessness of manner, with oscillation of the eyeballs, with tremor manifesting itself only on emotion, with paresis of the lower limbs, and finally with permanent contractions, we do not notice decided alienation of mind; there is nothing more than general enfeeblement and blunted emotional faculties.

Paralysis agitans may be confounded with general paralysis of the insane. But in paralysis agitans the voice is not really tremulous; there is rather a monotonous tone and uncertain utterance, which, with the fixed features, the sensation of excessive heat, the peculiar gait and attitude, the unaltered cutaneous sensibility, the tremor ever present except during sleep, and the very long duration of the symptoms, characterize the disease. The intellect becomes obscured towards the end of the malady, but not before.

Diseases characterized by Enlargement of the Head.

Chronic Hydrocephalus.—The signs of dropsy of the brain are progressive enlargement of the head, and a perversion or a gradual loss of one or several of the special senses, of the mental faculties, and of the power of voluntary motion. The child cannot bear the weight of the head; the gait is tottering and uncertain. The intellect, slowly but certainly, becomes deranged. As the malady advances, strabismus, partial palsies, epileptic convulsions, vomiting, cutaneous anæsthesia, and loss of sight, of smell, and of taste are observable; the bowels become very constipated; and a copious secretion of tears and of saliva is not infrequent.

Before death takes place, which sometimes does not happen for years, the child ordinarily becomes idiotic. A few cases recover; fewer reach adult age with the brain compressed by the accumulated fluid; in still fewer the disease does not develop until after childhood. If the patient survive until adult age, the size of the skull is

generally immense. I saw, some years since, a young man, twenty-two years of age, whose head measured fully two feet and a half in circumference. He could walk unaided, but often fell. He was half idiotic, and subject to epileptic fits; yet he had sufficient intelligence to understand what was said to him, and in his childish way to do as he was told.

Hydrocephalus may result from meningitis, from interference with the circulation through the veins of Galen, or from obstruction to the free movement of the cerebro-spinal fluid: occasionally no causative condition can be recognized.

The skull is sometimes very large without dropsy of the brain existing. The cranial bones may slowly thicken to an extraordinary degree from syphilis, or from unknown causes. The head may be overgrown, and its bones thickened and spongy, as in *rhachitis;* or it may be large when there is no disease. These states differ from chronic hydrocephalus by the absence of cerebral symptoms: and in doubtful cases we may resort to the ophthalmoscope as a means of diagnosis. The vessels of the eye, even in the early stages of chronic hydrocephalus, enlarge, and in proportion as the serum compresses the brain we find an increase of vascularity in the retina, with dilatation of its veins, and with an increase of the number of its vessels: complete or partial serous infiltration of the retina; and an atrophy, more or less perceptible, of the optic nerve. These lesions vary with the age of the disease and the amount of serous effusion; but none of them exist in rickets. Then in rickets the tendency is to spasm of the glottis, to diarrhœa,—and the head is rather square-shaped than globular. In very rare instances the size of the head has been observed to be increased in the cerebral palsies of children due to hemorrhage or embolism.

Hypertrophy of the Brain.—It is very questionable whether such a disease as a true hypertrophy of the brain exists. The enlargement, when not due to an unrecognized hydrocephalus, is mostly a congenital malformation, or is found in children in connection with rickets, with changes in the brain of a sclerotic kind, or with those alterations caused by a defect of brain substance to which the name porencephalus has been given, but where, at the same time, in other portions of the brain extensive cell infiltrations and connective tissue changes may happen. It is stated that, in hypertrophy of the brain, unlike hydrocephalus, when the fontanelles are touched, the sensation is that of a solid substance.

Diseases characterized by Enlargement of Various Parts.

Acromegalia.—In this peculiar and uncommon affection, first described by Marie, enlargement of the hands and feet occurs, as well as of the head, and especially of the face. Often bones and soft tissues both take part in the change, although the muscles may undergo wasting, with resulting weakness. The hands become broad and spade-like; the face assumes the shape of an elongated oval; the jaws, the malar bones, and the supraorbital arches are prominent; the forehead is receding. Spinal curvature is common. The disease occurs in young adults and pursues a chronic course; in rare instances it has been met with in children, especially in imbeciles. Changes in the pituitary body are constant; in some cases a tumor has been present, and in these headache, optic neuritis, and visual derangement have been observed. Somnolence, headache, and atrophy of the optic nerve are frequent. Sometimes changes in the thyroid gland have been noted, either enlargement or diminution in size. Occasionally dulness on percussion over the upper portion of the sternum has existed, and this has been attributed to persistence of the thymus gland. Rheumatic or neuralgic pains are not infrequent; and the tongue, lips, and nose may show striking increase in size, while the nails are small in proportion to the great growth of the bones in the hands and feet. Acromegalia may affect only one side of the body.

In *gigantism* there is symmetrical growth of all the bones and parts of the body, but there are neither ocular nor cerebral symptoms. Nor are there in *leontiasis*, in which the enlarged face is said to resemble that of a lion.

In *myxœdema* the tumefaction is not confined to the extremities, but is very general, and depends not upon changes in the bones, but upon a peculiar infiltration of the connective tissues. The skin is thickened and adherent to the subjacent tissues, and not pliable as in acromegalia. The face is "moon-shaped," and the jaws and malar bones are not projecting.

In the condition known as *osteitis deformans*, or *Paget's disease*, the changes in the bones of the face give to this the appearance of an inverted triangle. Besides, the disease attacks the long bones of the body, which undergo deformity; the spine curves; the face is not involved.

Certain chronic diseases of the lungs and pleura are attended with enlargement of the terminal phalanges of the fingers and toes, and of the distal epiphyses of the bones of the legs and forearms.

The finger-nails are curved, and the vertebral column is often bent. The changes are, however, usually restricted to these parts, and the disorder is not likely to be mistaken for acromegalia.

Diseases characterized by Paroxysmal Pain.

There is a group of nervous disorders characterized solely by pain, confined ordinarily to one nerve. These nervous pains bear the generic name of *neuralgia*. Indeed, in all neuralgias the chief symptoms of the disorder resolve themselves into one symptom,—the symptom of pain. The pains are acute, follow the course of a nerve-branch, and come on in paroxysms having distinct exacerbations, succeeded by distinct intermissions. In some cases these intermissions are long, in others short; in some they are complete, in others the pain is lasting and becomes from time to time exalted,—rather remissions, therefore, than intermissions. When the pain is severe it may be attended with muscular twitching. Sometimes, too, there is pallor followed by redness and swelling, though swelling is rare. Tenderness is present only when the neuralgia is of long continuance; at least there is not tenderness along the aching nerve, though we may find certain sensitive spots, as where a nerve-trunk emerges from a bony canal, or passes over a hard surface, or through fascia to become superficial, or at the point of division of a nerve-trunk, or of anastomosis of two nerve-trunks.

The pain of neuralgia is of a purely nervous character, and exists independently of inflammation, or of any recognizable textural change of the nerve-centres or nerve-trunks. Fixed pain and persistent early tenderness, evidences of lessened sensibility and of trophic changes in the skin or muscles, and cutaneous eruptions in the course of the affected nerve, bespeak *neuritis*, and not neuralgia. Indeed, it is only when, after a minute search, we can detect no definite organic cause for the local pain, that we may conclude that our patient is laboring under neuralgia. Among other points of difference, too, we observe that the pain of neuralgia is often relieved by deep pressure, while that of neuritis is thus increased, as it is by movement; that it intermits much more completely; and that in neuritis we often have the history of contusion or strain, or of extension of inflammation from parts near by. Changes in the hair may take place as the consequence of either neuritis or of neuralgia.

From the characteristics of the pain just mentioned, it is evident that it is not likely to be confounded with that of ordinary local inflammation. But there is a kind of local pain for which neuralgia is often mistaken: the pain of *subacute* or of *chronic rheumatism*. Yet

this is in reality very dissimilar. The rheumatic pain is attended with soreness, is aggravated by movement or by pressure, is more diffuse and irregular, much more constant, much more influenced by alternations of temperature, but not acute or paroxysmal, and not limited anatomically to the course of a nerve, but scattered over parts supplied by several. In studying the relations of rheumatism to localized pain we must bear in mind that exposure to cold is also a frequent cause of neuritis, which then appears to have a rheumatic origin. Except as regards the influence of weather, the pain of *myalgia* presents much the same points of difference as the pain of rheumatism, in addition often to the history of a muscular strain.

The source of the neuralgia should always be determined as closely as possible, on account both of the prognosis and of the treatment. In many cases it will be found to be connected with anæmia; in others, with the poison of rheumatism, of lithæmia or gout, of malaria, of syphilis, or of uræmia or ptomaines, or to be due to injuries to nerves by contusion or wounds. It may be owing to emotional disturbances, to neurasthenia, or to exposure to cold. It is often reflex, the pain being far away from the seat of the disease. For instance, an affection of the digestive apparatus, of the liver, or of the kidneys, may give rise to neuralgia in parts quite remote from them.

Neuralgia may occur in any portion of the body. It may shift rapidly from one part to another, as in that peculiar neuralgia described by Putegnat,[1] excited by a desire to pass water and by the act of micturition, beginning with numbness and acute burning or lancinating pain along the urinary passages, then affecting particularly the nerves of the forearm, especially the ulnar, and disappearing completely after micturition. The most frequent seat of neuralgia is about the head; and we shall here notice chiefly a few of its most common kinds. The other varieties of the disorder will be elsewhere alluded to.

Facial Neuralgia.—The sensory branches of the fifth pair are often the site of agonizing pain. But all the branches of the nerve are not equally liable: the lowermost of them is rarely affected. When the supraorbital division is the seat of the ailment, the pain shoots to the forehead, the eyebrow, and the eyeball, which is apt to become injected. There are tender points just above the supraorbital notch or foramen, in the outer part of the upper eyelid, at the lower edge of the nasal bone, and sometimes within the eyeball. If

[1] Gazette Hebdom. de Méd. et de Chir., April, 1864.

the infraorbital nerve be disturbed, the pain darts to the upper lip, to the upper row of teeth and the posterior nares, and the cheek reddens and tingles, or the eyelids twitch. Tender points are found at the infraorbital foramen, at the side of the nose, over the malar bone, and on the gums of the upper jaw. When the pain occurs in the inferior branch, it radiates to the lower lip and the chin, and is frequently accompanied by a flow of saliva. Tenderness exists at the inferior dental foramen, on the temple, and over the parietal eminence. Sometimes only one, at other times two, at other times all of the branches of the fifth are implicated in the complaint, or they may be seized upon alternately. There is often also pain at the vertex.

The disease is one of those belonging to advancing years; one of the neuralgias of bodily decay on which Anstie dwells. It has the same general causes as any other form of neuralgia. Sometimes it is associated with decayed teeth, or with an abnormal state of the bones of the head or face, such as thickening of the frontal, ethmoid, and sphenoid bones. Many of these cases terminate, after months or years of excruciating agony, in apoplexy.[1] When from decayed teeth, the pain finally localizes itself in the dental arch, and there is persistent discomfort in addition to the neuralgic exacerbations.[2]

The intervals between the paroxysms of neuralgia are of varying length. They may be of six months', or even a year's, duration; but so long an intermission is uncommon. Sudden changes of weather generally excite attacks.

The malady is easily recognized. The pain from disease of the bones of the face may be mistaken for it. But this pain is not paroxysmal. *Painful anæsthesia of the fifth nerve* is discriminated by the insensibility of the painful portions to touch, or indeed to any irritation. *Spasm of the face* is distinguished by the absence of pain from the convulsive twitchings of reflex origin which sometimes take place in facial neuralgia or "tic douloureux."

The *epileptiform neuralgia* described by Trousseau is dissimilar in these peculiarities: whether simple or combined with rapid convulsive movements of the muscles on one side of the face, it is quickly over; it lasts but ten or twenty seconds at a time, never more than a minute. Yet during the short duration of the seizures the pain reaches an intensity greater than in ordinary neuralgia. Moreover, in some

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[1] Sir Henry Halford's Essays and Orations, p. 37 *et seq.*

[2] An interesting collection of cases is given in an essay by Brubaker on Reflex Neurosis associated with Dental Pathology.

persons who suffer from this terrible malady—the attacks of which may happen in quick succession by day as well as by night, and then perhaps remit for weeks or months—vertiginous sensations or epileptic fits occur, and thus the diagnosis is facilitated by the history of the case.

Hemicrania.—The pain here is limited to one side of the head, but it may extend to the other side, and be bilateral. It is intensified by sound of any kind, by movement and by light, and is often preceded by disorder of sight,[1] sometimes of hearing and taste, by numbness and tingling in the limbs, by transient aphasia, by a sense of weight, though rarely by muscular weakness. Nausea generally attends the headache, but is most pronounced when this has reached its height, and is, as a rule, followed by vomiting or retching; the nausea and vomiting of the "sick-headache" are usually, indeed, prominent features of the paroxysm, hardly less prominent than the pain. During the attack there is commonly pallor of the face, which at the close gives place to flushing; slight mental change may also be noticeable. The attack lasts for hours or days; often it is severe for half a day. It may end with free diaphoresis or diuresis. At its termination, the patient feels exhausted, yet soon recovers his usual health, and may remain free from a seizure for a long time. But, as the disorder commonly happens in women at their menstrual periods, the interval is not apt to extend beyond four weeks. At times the sensory phenomena of migraine occur without headache.

Hemicrania, migraine, or megrim, has been explained as a neurosis of the sympathetic; or as a discharge of nerve-force, a "nerve-storm," from centric disorder. It is a stubborn affection, the tendency to which diminishes after middle age, but which, as Liveing[2] clearly demonstrates, has an hereditary character.

Hemicrania must be carefully separated from the pain in the head that accompanies an *organic cerebral affection*. The main points of distinction are, that the neuralgic malady is paroxysmal, is attended with the same group of symptoms during each attack, and produces no nervous derangement in the intervals between the seizures.

Rheumatism of the scalp differs from hemicrania in the pain being continuous, dull, and superficial; in occupying generally both sides of the head; in being augmented by moving the affected muscles, and

[1] There may be obliteration of objects in the field of view, or a curious glimmering attended with colored outline near the outside corner of the field of vision. These ophthalmic migraines have been described by Charcot (vol. iii. of his Clinical Lectures) as being at times among the forerunners of general paralysis.

[2] On Megrim, London, 1873.

relieved by warmth. Moreover, there is almost always other evidence of rheumatism, and the pain is intensified by pressure; whereas in hemicrania, although the hair may be sensitive to the touch, strong pressure on the forehead, and even on the hairy part of the scalp, does not increase the pain, may, indeed, afford relief.

In *periostitis* affecting the bones of the head, particularly when syphilitic, we may find the same violent pain as in hemicrania. But there is considerable tenderness on pressure, the parts attacked are swollen and less elastic than the healthy portions, and the pain is especially severe at night.

Sciatica.—This is sometimes a neuralgia following the course of the sciatic nerve, but often it is a neuritis. The seat of the greatest suffering is generally the lateral surface of the thigh; thence the pains extend to the popliteal space, and in some instances along the anterior part of the leg. Often, too, the patient complains of an aching near the sciatic notch and in the loins. The pain is more or less steady; but it has its periods of fierce exacerbation, and damp, cold, and pressure augment it. When the nerve is inflamed there is tenderness on pressure over the course of the nerve. Pressure on localized points always develops pain, and the points that are most marked are on the lower end of the sacrum, on the side of the trochanter opposite the emergence of the great and small sciatic nerves, various points on the posterior aspect of the thigh, one at the head of the fibula, and one behind the outer ankle.

The disease is obstinate, and lasts for weeks or months. It interferes with locomotion, because of the distress which movements of the leg and foot occasion. It is much more frequent in men than in women, and is a very rare disease in children. Generally it depends upon exposure to cold, or upon the rheumatic diathesis, or upon a neuralgic predisposition, or upon an irritation affecting the nerve before it leaves the pelvis, the result not unusually of sexual disorder, or of pressure from a gravid womb, or from an accumulation of fæces in the lower bowel. In many instances it is connected with gout, in others with anæmia, with syphilis, with disease of the hip-joints, and it may be, although it very rarely is, symptomatic of cerebral disease. Occasionally it is due to reflex excitation of the nerve. Sometimes it occurs after forced marches or long rides; probably in many of these cases, however, the sciatica is rheumatic. It is seldom double, except when of diabetic origin, or when due to compression from a growing tumor in the pelvis or from enlarging cancerous vertebræ.

Sciatica, when of long duration, leads to loss of motor power in

the leg, to tingling, and to anæsthesia; and certain nutritive changes are observed in the limb, which is found to have dwindled, or there may be œdema. When the disorder is the result of neuritis, there is generally decided and persistent tenderness,—in pure neuralgia there is not much,—and movement and position have marked influence on the pain. Further, the history of the case in pure neuralgia, the spontaneous pain, the usual anæmia, and the previous occurrence of, or the coexistence with, other neuralgias, are very significant. Occasionally the neuritis ascends to the cord.

An effusion within the sheath of the nerve may, according to Fuller, be inferred when a patient who is suffering from sciatica complains of a dull aching or a benumbing pain in the limb, causing it to feel swollen, and when this sense of numbness and increased bulk has succeeded to pain of greater intensity, accompanied by cramps and startings and more or less inability to move the limb.

The disorders which are most likely to be confounded with sciatica are: *rheumatism of the muscles* and fibrous sheaths around the hip-joint; affections of the joint; and pains caused by irritation of the kidney. The first is very readily distinguished. It is generally, what sciatica is rarely, double-sided; and the pain is dull, diffuse, not paroxysmal, not limited to the sciatic nerve and its area of distribution, nor as much increased on pressure as that of sciatica. But, practically speaking, this kind of rheumatism is seldom seen unless associated with rheumatic inflammation of the sciatic nerve.

In *affections of the hip-joint* the suffering is increased by standing with the weight of the body thrown on the diseased leg. Moreover, the pain does not descend in the course of the sciatic; is not associated with tenderness of the nerve; the aspect of the limb points to the disorganization that is going on; the leg shortens. Yet, before admitting this as a mark of difference, it must be ascertained by careful measurement; for, in consequence of muscular contractions, the affected limb in sciatica may appear to be shorter than it is. The main points of distinction between sciatica and a nervous affection of the hip-joint are the usual combination of the latter with hysteria, the very superficial tenderness, and the fact that the pain is apt to extend over the whole thigh.

Irritation of the kidney causes pain shooting down the thigh. The distress exists, however, in the course of the anterior crural nerve, is therefore not localized in the sciatic, is unassociated with tenderness, but is accompanied by a frequent desire to pass water, and by other signs of disorder of the urinary function.

Sciatica is sometimes *feigned*, especially by soldiers. But the copy

is rarely a very accurate one. Impostors complain of pain on pressure and on motion, but are ignorant that the pain is prone to exacerbate after intervals of comparative quiet, and to increase in violence as night approaches. Their fancied torment is constant, but does not prevent them from sleeping; they wince when the muscles of the thigh are touched, yet, if their attention be diverted, the hand may be pressed along the sciatic nerve without any sign of tenderness being manifested.

General Crural Neuritis.—In this disease, much rarer than sciatica, there is extensive inflammation of the fibrous sheaths covering the lumbar and sacral plexus; in consequence many of the nerves of the leg are involved at their origin, and there are signs of widespread neuritis. There is pain along the course of several nerves, and motion is somewhat impaired, and there may be muscular atrophy; the tenderness of the nerve-trunks is most apt to be found near the pelvis, and this is an important sign as distinguishing the complaint from disease of the spinal cord. The pain is sometimes reflected to the sound side. The knee-jerk is usually increased. The disease occurs mainly in gouty or rheumatic persons, and is apt to be of considerable duration. It may affect pre-eminently a single nerve, as the anterior crural; and the sensory phenomena, especially anæsthesia on the front of the thigh, are then very marked.

Brachial Neuritis.—This is a rare and very perplexing form of neuritis; more strictly speaking, it is usually a perineuritis,—a primary inflammation of the sheaths of the branches that form the brachial plexus. It is a disease of the latter part of life, met with chiefly in the rheumatic or gouty. The pain is very great, and comes on in paroxysms; but, irrespective of these, a dull pain or ache is constantly present. The pain has its seat above the clavicle, in the axilla, in the region of the scapula, and the inner part of the shoulder-joint. It lancinates to the neck and chest, and sometimes along the course of the arm, giving rise there to a sense of weight and heat. Motion will induce the pain, even walking may. There is sensitiveness of the skin near the affected part, and flabbiness and slight wasting of groups of muscles, which may even prevent the reaction of degeneration; over the atrophied muscles anæsthesia is at times met with. There may be persistent tenderness of the nerves near their origin, but this is not always easy to determine; the influence of movement in evoking pain is always striking and almost immediate.

During the paroxysms of pain, which are most apt to come on in the latter part of the day, when fatigued, there is a sense of constriction at the upper part of the chest with some shortness of breathing,

and in consequence the disease, when left-sided,—and it happens that all the cases I have seen have been so,—is apt to be mistaken for *angina pectoris*. This occurs the more readily since both affections are diseases of advancing years, and there may be coincident degenerative changes in heart or arteries; and irregular heart action is not an unusual attendant. The great difference, besides the exact seat of pain, is that in brachial neuritis some pain or tenderness is always present, and always intensified by movement, and that we do not have the rapid appearance and disappearance of the agonizing paroxysms that distinguish angina; the local changes in the muscles in brachial neuritis are also of value. These, too, help us, in addition to the persistent tenderness and the influence of motion on the parts, in distinguishing the cases of pure brachial neuralgia from brachial neuritis. In some instances, in place of rheumatism or gout causing the neuritis, we observe it after contusions or dislocations of the shoulder, or from the pressure of enlarged glands or tumors. In any case, owing chiefly to the constancy with which the arm is kept quiet, fixity of joint and arthritic changes may supervene.

There is a form of rheumatism in which the interstitial tissue of the nerves and muscles is affected, to which Gowers has given the name of *neuromyositis*, which very closely resembles brachial neuritis, with which, indeed, it may be combined. Here the mere expectation of movement produces pain, as do passive movements and compression of the nerve-endings by voluntary contractions; the muscles are tender, and the joints, usually the shoulder-joint, may become fixed and the seat of adhesions, and add to the rigidity and the inhibition of movement. The pain in neuromyositis is altogether connected with motion or the expectation of motion, and there are no paroxysms of spontaneous pain, as is so marked a feature of brachial neuritis.

CHAPTER III.

DISEASES OF THE UPPER AIR-PASSAGES.

SECTION I.

DISEASES OF THE NOSE AND ASSOCIATE ORGANS.

THE nasal chambers, pharynx, larynx, and trachea constitute the upper air-passages. As the disorders of the nose and the naso-pharynx, or the space between the plane of the posterior nares and a horizontal line drawn through the lower end of the soft palate, belong largely to a class which requires surgical treatment, a brief review will be given in this place of those only that have features of medical interest. The *frontal sinuses* are in direct connection with the nasal chambers, and, in case of occlusion of their normal outlet, there is an accumulation of secretion, which may cause *headache*. This is likely to occur especially in the catarrhal inflammation attending influenza, in which the headache may continue for weeks until the inflammation subsides, or the patency of the outlet is restored. The headache may be associated with vertigo. Tumors may develop from the mucous membrane lining the frontal sinuses, of the same character as those of the nasal chambers; and the larvæ of insects, or mature forms, such as centipedes, find their way at times into these cavities and there cause pain and irritation.

Frontal sinus diseases are to be distinguished from *supraorbital neuralgia*, *migraine*, and *cerebral disease* by careful inspection of the interior of the nose, by examination with the curved probe to test the openness of the canal, by the presence of tenderness and other local signs of inflammation, such as swelling or discoloration, and by the history of the case.

The *affections of the antrum Highmorianum*, or maxillary sinus, are similar to those of the frontal sinuses, and are more surgical than medical. Many of these cases are attended by a pain *above* the corresponding eye, and may be mistaken for *migraine* and the *neuralgia of frontal sinus disease*.

The diseases of the nasal chambers may be divided into acute and chronic. Among the former are coryza, acute rhinitis, hay-fever, hemorrhage or epistaxis, hydrorrhœa, mycosis, and abscess. Promi-

nent among the second class are rhinitis, hypertrophic and atrophic, cirrhosis of the mucous membrane, ethmoiditis, thickening and deviation of the septum, rhinoscleroma, new growths, specific destruction by tuberculosis, syphilis, lupus, and malignant disease.

Another division might be made into local affections and those occurring in eruptive fevers and other acute diseases. For instance, in scarlatina, smallpox, typhoid fever, and diphtheria, swelling with increase of secretion and ulceration may occur, while in rheumatism and influenza peculiar changes are noted. Many, probably most, of the instances of marked deviation of the septum and associated abnormalities are not evidences of disease, but are due to former fractures with unreduced dislocation, or to heredity. Bryson Delavan has found asymmetry in the nasal chambers to be the rule rather than the exception.

As regards affections of the nose attended by mucous or purulent discharge and more or less obstructed breathing, it is well to bear in mind the possibility in children of there being a *foreign body in the nose*, and in older patients *rhinoliths* are sometimes detected upon rhinoscopic examination. In one remarkable case described by Warren,[1] the breech-pin of a gun was discovered embedded in the right nasal fossa, where it had been driven by the explosion of a gun, several months previously, and its presence in the nose had not been suspected.

Coryza, or *acute catarrh*, is a general affection, which manifests itself by inflammation of the mucous membranes of the nose especially, but other mucous surfaces of the air-passages may be previously or subsequently affected. It often follows exposure to cold and dampness, and attacks those principally of lowered vitality. *Rhinitis* may be due to local irritation, as rough manipulation, operations upon the nose, or strong applications. It may be confined to one nostril. Upon inspection, the mucous surface is of a bright red color in certain locations, the turbinated erectile tissue is engorged, shows abrasions, and bleeds readily. The fossa may be obstructed to a greater or less degree by inflammatory swelling of the mucous membrane, and the secretions vary in density from a clear serous fluid to a caseous or fibrinous exudation. To the latter the names of *rhinitis caseosa* and *rhinitis fibrinosa* have been applied. Cases with marked œdema have been reported under the title of *rhinitis œdematosa*.[2]

Diphtheria of the nose may occur independently, but is usually a concomitant or sequel of faucial diphtheria. At the present day the

[1] Surgical Observations, Boston, 1867.

[2] J. C. Mulhall, Trans. Am. Laryng. Assoc., 1893.

presence of the Klebs-Loeffler bacillus is relied upon to decide the diagnosis of diphtheria, although this micro-organism has occasionally been found in affections of the throat and nose, which give no clinical evidence of being diphtheritic, and it is present in many cases with purulent discharge from the nose. An acute catarrh is sometimes an early symptom of some specific disease, of *measles*, for instance. Nasal catarrh accompanies *erysipelas* and *influenza*. Nasal catarrh may be also produced by the administration of remedies, as of the iodides, or, by the inhalation of drugs, such as ipecacuanha. At certain times of the year, when the air is filled with pollen from the artemisia absinthifolium, or ragweed, the ailanthus tree, or from roses or grasses, many persons suffer from what is called *hay-fever*, hay-asthma, rose-cold or hyperæsthetic rhinitis, which has for its most marked sign the reappearance of the symptoms upon the same day each year, suggesting a strong neurotic element. Daly, Roe, and Sajous have shown the dependence of many of these cases upon nasal abnormalities. A severe purulent rhinitis of acute form may be caused by accidental *gonorrhœal infection of the nose;* the history of the case gives the explanation, and the discovery of the *gonococcus* the demonstration.

Nasal hydrorrhœa is distinguished from *coryza* by the excessive flow of a serous fluid from the mucous membranes, especially over the turbinated bodies, which are pale and sodden. The affection is unilateral, but it may be bilateral. Unlike hay-fever, it happens at all seasons, though, like hay-fever, it is found in neurotic subjects. It may occur in paralysis of the trifacial nerve, or as a result of head injury, and may be associated with polypi, or myxoma of the nose; it is at times of months' duration.[1] *Arteriosclerosis* affecting the vessels of the mucous membrane of the nose is evidenced by a special disposition to coryza and pharyngeal catarrh, with local congestions and tendency to nosebleed. Such patients are especially liable to obstruction of the nasal passages by temporary swellings which are followed by free effusion of watery secretion.[2] In *cerebro-spinal rhinorrhœa* there is constant dripping from the nose of a fluid of specific gravity about 1005 from which mucus and proteids are absent, and which on boiling reduces Fehling's solution.[3]

Nasal hemorrhage may indicate a general condition of the vascular

[1] As in a case reported by C. E. Bean before the Laryngological Association in 1891.

[2] James T. Whittaker, Pennsylvania Medical Journal, Feb. 1899, p. 459.

[3] St. Clair Thomson on Cerebro-Spinal Rhinorrhœa, 1899.

system, a degeneration of the arterial coats, with or without increased tension. Occurring after the middle period of life, in a person otherwise apparently healthy, it suggests the likelihood of apoplexy or contracted kidney. It is important to distinguish between those cases in which the blood only passes through the nose, and those in which the blood comes from the nose. A rhinoscopic examination, both anterior and posterior, is essential to an exact diagnosis. In nasal hemorrhage the blood very frequently comes from the septum low down, where it can be easily inspected.

Post-nasal catarrh has for its prominent symptoms the dropping of mucus from the soft palate into the throat, and the expulsion, usually in the morning, of masses of gelatinous mucus or of hardened crusts from the naso-pharynx, giving rise to the unpleasant habit of hawking and spitting. In such cases rhinoscopic examination reveals inflammation of the mucous membrane in the vault of the pharynx, and the glandular tissue, or so-called "pharyngeal tonsil," may be hypertrophied and form polypoid excrescences, or large adenoid tumors that may entirely occlude the posterior nasal openings. Digital exploration is a valuable means of diagnosis, especially in children. Deafness may be caused by occlusion of the Eustachian tubes, and mouth-breathing is a necessary sequence. Snoring, dryness of the throat, and night-terrors occurring in children, may also be due to this condition. A form of post-nasal catarrh, of less severity, attends posterior hypertrophies and other abnormalities of the nose attended by increase of secretion. The diagnosis is made by rhinoscopy.

In *glanders* the purulent or sanious discharge from the nose is attended with erysipelatous blush on the nose and cheeks, characteristic pustules on the face and in the nasal passages, and the symptoms of pyæmia.

The chronic forms of rhinitis accompanied by catarrhal thickening of the septum or of the turbinate bodies, atrophy or cirrhosis of the mucous membrane, and the development of cysts, polypi, or papillary fibromata, are recognized by careful rhinoscopic examination, and belong to surgery rather than to medicine. In former times ozæna was regarded as a disease, but it now is recognized as an attendant upon chronic atrophic rhinitis, in which the secretions dry into crusts which undergo putrefactive changes, and thus produce the offensive odor. In cirrhosis there are contraction of the mucous membrane and evidences of atrophy, without decided catarrhal symptoms, thus showing a constitutional origin. Hypertrophy of the mucous membrane is considered as a preliminary stage to atrophy; and the production of polypi, or of myxoma, or, more rarely, of fibroma, is not

an uncommon result. Hypertrophies are distinguished from new growths by their situation, color, density, and immobility; new growths being in abnormal situations, of peculiar color, and pedunculated; for instance, *polypi* are white and glistening, *cysts* are white, *papilloma* may look like a small bunch of grapes. The diagnosis of papillary hypertrophy from true papilloma, papillary fibroma, depends upon its location, appearance, and the microscopic details, which also distinguish the latter from epithelioma and sarcoma. Abscess most frequently appears in the septum, when it occurs in the nose.

In making the diagnosis between benign and malignant growths, the rapid development and general appearances of the latter are usually depended upon as conclusive. At the same time the difficulty is enhanced by the danger of benign growths becoming transformed in their character. Bosworth reports a case in which sarcoma developed after polypi had been operated upon rather harshly by means of forceps, and a similar case is narrated by Heyman.[1] Traumatism has been also observed to result in fibrosarcoma and other malignant growths of the nasal chambers.

The reflex disorders arising from naso-pharyngeal obstruction, by hypertrophies or new growths, have been studied by a multitude of clinical observers. Obstinate headache, asthenopia, earache, persistent cough, and vertigo are symptoms of special cases of nasal disorder. Voltolini first directed attention to the fact that nasal polypi may be the cause of *asthma*, and a deflected septum and hypertrophic rhinitis are also claimed as causes by Bosworth. Weber showed that diseases in the upper air-passages readily produce turgescence and swelling of the bronchial mucous membrane. Glycosuria has been known to originate in nasal obstruction, and to disappear when this was removed.[2] *Nasal polypi* apparently may cause spasmodic stricture and difficulty in urinating, as in a case reported by Mulhall;[3] the stricture was at once relieved by the removal of the nasal polypi.

Rhinoscleroma is a form of new growth allied to round-celled sarcoma, characterized by the appearance of flat, slightly raised patches which are smooth on the surface and of ivory-like hardness, and which first appear at the edges of the nostril, spread to the upper lip, and do not ulcerate. Von Frisch discovered in the growths little bacilli, resembling the pneumococcus of Friedlander, and like the latter encapsulated, but differing from it in its response

[1] Revue Mensuelle de Laryngologie, 1888, p. 24.

[2] Bayer, Revue de Laryngologie, 1894, xv. 19.

[3] American Laryngological Association, 1892, p. 42.

to staining by Gram's method. Inoculation has failed thus far to reproduce the disease in the lower animals.[1] The lesion may gradually extend into the tissues of the mouth and nose and to the larynx. Rhinoscleroma is to be distinguished from syphilis, epithelial cancer, and keloid. It differs from venereal disease mainly by its very chronic course, the absence of softening or ulceration, and its absolute intractability under every kind of medication. From epithelioma it can be discriminated by its smooth, glistening surface, its hardness, the absence of bleeding or ulceration, and its persistently local character. The history of the case and its general appearance distinguish it from keloid, which has the puckered, white, and irregular outline of scar-tissue.

SECTION II.

DISEASES OF THE LARYNX AND TRACHEA.

Of these affections those of the larynx are far the most frequent and the most readily recognized. There are, indeed, symptoms in laryngeal diseases which at once direct attention to the seat of the malady. The larynx is the organ of speech: hence changes in the *voice* constitute the most striking manifestations of disorder. These changes vary in degree. The voice may be merely hoarse or completely lost. In young children the different tone of the cry corresponds to the altered voice of adults. The alteration of the voice depends almost wholly upon an affection of the vocal cords, and this may be organic, such as inflammation, œdema, ulceration, cicatrices, and morbid growths; or it may proceed from perverted or impaired innervation. Very often the hoarseness or loss of voice is caused by diminished tension and want of certain and prompt action of the vocal cords, whether connected with structural change or not. The same cause gives rise, for the most part, to the modifications of the voice, which show themselves as huskiness in speaking, or in the loss of certain notes in singing.

Next to the voice in diagnostic importance stand the character of the breathing and the cough. The *breathing* is labored and difficult, and is frequently perceived to be noisy, and coarse or shrill,—the so-called *laryngeal stridor:* a sign encountered whenever the orifice through which the air has to pass is narrowed, either temporarily by a spasm, or more permanently by any state which gives rise to a constriction of the parts; for instance, by swelling of the mucous membrane, or diphtheritic deposit.

[1] Text-Book upon the Pathogenic Bacteria, by Jos. McFarland, Philadelphia, 1898.

The difficulty in breathing is in some cases slight; in others great. One of the peculiarities of laryngeal dyspnœa is its tendency to recur in paroxysms, during which the patient appears to be in imminent danger of strangling. These fits of suffocation are produced mostly by a spasm of the intrinsic muscles, particularly the adductors of the larynx. Attacks of dyspnœa also are met with in cases of paralysis of the abductors of the larynx, or paralysis of the posterior crico-arytenoid muscles. The attacks occur in pure spasm of the glottis; in croup; in œdema of the larynx; in ulceration and in polypi of the larynx.

The *cough* of laryngeal affections presents frequently the same peculiarity as the dyspnœa,—it happens in paroxysms. Another peculiarity, although not one so constant, is its harsh and ringing tone. The cough is often short and dry; sometimes it is followed by muco-purulent expectoration of roundish shape, or by a blood-streaked sputum, or by the spitting up of false membrane. It is readily excited by the act of swallowing, its seat is referred by the patient himself to the windpipe, and is especially troublesome at night.

Pain is not so unusual a symptom of laryngeal disease as either cough or changed breathing. In chronic affections it may be, indeed, wanting. It is rarely severe; often more a sensation of tickling, of burning, or of uneasiness than of actual pain. It is apt to extend down the trachea to the upper part of the sternum. Sometimes it is increased on pressure, as in acute laryngitis and in ulceration of the mucous membrane; and it may be also augmented by the act of swallowing.

By the symptoms, then, of altered voice, cough, dyspnœa, and, in some cases, of local pain and difficulty in deglutition, we recognize a laryngeal affection. But to do so with accuracy, the larynx must be inspected with the laryngoscope. It may be either circular, square, or oval. The circular mirror occasions least irritation. The larger the mirror we can employ, the better is the image.

The mirror is in some cases all that is necessary to practise laryngoscopy. It is heated in warm water or over a lamp and then introduced into the back of the mouth in the manner presently to be described; the person to be examined having been placed with his face towards the sunlight, so that its rays may strike the laryngeal mirror.

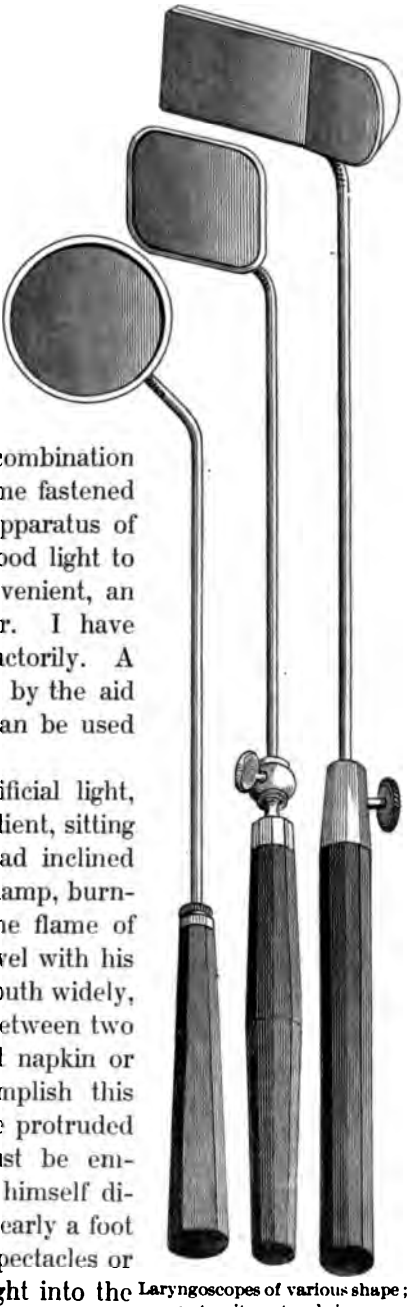
But examinations by direct light are practicable only on clear days and at certain periods of the day. Usually we require a second mirror to illuminate the throat and the laryngoscope. This mirror is of circular form, about three inches and a half in diameter, and with

a focus of about fourteen inches. It may be either attached to the head by means of a band, or worn on a pair of spectacle-frames, or placed on a movable stand, or affixed to a lamp. When the frontal band is made use of, the observer may either place the mirror opposite to one of his eyes, and look through the central perforation, or adopt the easier method of wearing the reflector on his forehead.

The light may be concentrated directly into the throat by a lens or a bull's-eye condenser, or by a combination of lenses attached to a metallic frame fastened to a lamp, as in the well-known apparatus of Tobold numerously modified. A good light to employ is coal oil; the most convenient, an argand or a Welsbach gas-burner. I have used the electric light very satisfactorily. A portable electric light is obtainable by the aid of a small storage battery, which can be used at the bedside.

To examine the larynx by artificial light, we should proceed thus. The patient, sitting in an upright position, with his head inclined slightly backward, is placed near a lamp, burning with a steady, brilliant light, the flame of which is behind and about on a level with his eyes. He is directed to open his mouth widely, to put out his tongue, and to hold between two fingers its point enveloped in a soft napkin or handkerchief. If he cannot accomplish this readily, the examiner must hold the protruded tongue, or a tongue-depressor must be employed. The observer now seats himself directly in front of the patient, and nearly a foot from the mouth. Putting on his spectacles or frontal band, he throws a disk of light into the back part of the mouth; he then rapidly intro-

FIG. 15.



Laryngoscopes of various shape; not quite natural size.

duces the laryngeal mirror, previously heated in warm water or over a lamp and its proper temperature ascertained by touching his own hand or cheek. The mirror, great care being taken not to bring it in contact with the tongue, is placed with its back against the uvula, which, with the soft palate, is pressed backward and upward; the lower surface of the laryngoscope should be firmly applied to, or, if this be found to occasion too much irritation, should be held near, the posterior wall of the pharynx. The inclination of the mirror varies with the position of the patient and the parts we wish particularly to explore. As a general rule, it may rest at an angle of about forty-five degrees.

Fig. 16.

Laryngoscopic examination, as made with the reflector attached to a spectacle-frame.

When one of the ordinary stationary laryngoscopic lamps is employed, the reflector is attached to the lamp by a freely movable brass rod, and the light concentrated on it is thus thrown into the mouth. In the laryngeal mirror the image is readily perceived. We see the epiglottis, the glottis, the cartilages, the true vocal cords, the superior thyro-arytenoid ligaments or ventricular bands, and in some cases even the rings of the trachea. We may be able to discern each

portion of the laryngeal aperture with distinctness, or it may take several examinations to do so.

In health, the color of the various parts is very different. Stoerck has well described it in likening that of the epiglottis, the interior of the larynx below the glottis, and of the cricoid cartilage, to the coloration of the conjunctiva of the eyelid; and the hue of the aryepiglottidean folds and the prominences of the arytenoid cartilages to that of the gums. The mucous membrane of the trachea between the rings is of a pale pink color; the vocal cords have a white, glistening look. Mackenzie takes special notice of the whole of the under surface of the epiglottis being in some cases of a bright-red hue; and Gibb points out that in negroes the cartilages of Wrisberg have a yellowish tinge.

FIG. 17.

Laryngeal image, as seen in the laryngoscope under favorable circumstances.

The laryngeal image in the mirror bears this relation to the real position of the parts: the right vocal cord of the person who is examined is seen on the left side of the mirror, and the left vocal cord on the right; or, to state the matter in a form easy to be remembered, the cord which corresponds to the right hand of the patient is the right, that seen towards his left hand is the left. The epiglottis appears in the laryngoscope at the upper portion and behind; so do the other structures that lie in front. The arytenoid cartilages show at its lower portion, and towards the front.

To judge of the movements of the vocal cords, we tell the patient alternately to inspire deeply and to utter, as a prolonged high note, a sound like "ah." During this the vocal cords are closely approximated and stretched, and the epiglottis, in fact the whole larynx, is somewhat elevated; while during a full inspiration the cords are far apart, and hence the glottis is wide open. To obtain a satisfactory sight of the deeper-seated parts, we must bear in mind that the more horizontally the surface of the mirror is placed, the more distinctly they come into view. For the exploration of these structures, and particularly of the trachea, the light must be thrown from below upward upon the laryngoscope. To elevate the larynx decidedly, and especially to bring the epiglottis fully into view, the patient should in a high pitch pronounce *ee* as in the word *see*.

In some, laryngoscopy is easy; a conclusive examination may be

made at the first attempt. In others, a course of training is required to subdue the sensibility of the fauces, which may be general, or be limited to a very small spot. As a means of overcoming the difficulty, sucking small pieces of ice, or the previous administration of bromide of potassium, or the local use of a solution of cocaine from two to five per cent., is useful. But the best means is skill in the use of the instrument,—its rapid and decisive handling. The administration of an anæsthetic may be, however, necessary. This is especially the case in refractory children suffering with papilloma or other conditions demanding ocular inspection. To overcome pharyngeal and laryngeal reflexes, Scanes Spicer[1] recommends the cautious use of a ten per cent. spray of solution of cocaine, and, for removal of salivary secretions, the free use of dry mops of absorbent cotton-wool.

In some persons with very irritable throats, I have obtained good views by pressing the instrument against the roof of the mouth, instead of passing it back into the pharynx, and by altering the position of the head a little, tilting it more backward. The epiglottis, and the structures at the entrance of the windpipe, are thus readily enough brought into view: with the deeper parts we do not succeed so well; but in many cases we get sufficient guide for topical applications. There are further obstacles, such as a rising up of the tongue, greatly enlarged tonsils, a long uvula, a pendent epiglottis, all of which at times interfere with our investigations. But in any case we should not endeavor to make the view more satisfactory by constantly altering the position of the mirror. It is better to introduce it repeatedly than to shift it often when introduced, or to keep it for any length of time in the patient's mouth. Digital examination of the larynx with the index-finger is an expedient of value in children and others who will not permit laryngoscopy. It is practised for diagnostic purposes in laryngeal œdema, and in new growths, such as papilloma.

Direct examination of the larynx can be made in a certain proportion of cases by Kirstein's instrument, which is a modified tongue-depressor connected with an electroscope. The epiglottis and pharynx having been painted with cocaine, or the patient moderately anæsthetized, the instrument is introduced into the mouth so that by its aid the base of the tongue may be pulled strongly forward, and the larynx be brought directly in line with the eye of the physician. This method is also useful to aid in the removal of new growths or foreign bodies. Waggett has shown how the X-rays may be utilized to locate foreign bodies in the larynx and œsophagus, and to determine the

[1] Journal of Laryngology, Rhinology, and Otology, London, Oct. 1894.

relative position of a probe and the foreign body. Some new growths may also be detected in this manner.[1]

If the mirror be passed behind the uvula, and the reflecting surface directed upward, the posterior nares may be examined. To practise *rhinoscopy*, however, the mirror should be small and fixed to the shaft at a right angle. The patient is directed to keep his head erect, or bend it slightly forward, and while his mouth is wide open a strong light is thrown to the back of the throat. But before the rhinal mirror is placed in position, a tongue-depressor is applied, with which the back of the tongue is well pressed down, and which may be given to the patient to hold. To get the uvula out of the way, a palate-hook may be used, by which means the uvula, with a portion of the soft palate, is gently drawn forward and upward, the handle of the hook being held to one side of the mouth: Voltolini's palate-hook widens the pharyngo-nasal space satisfactorily, or Sajous's soft palate-elevator may be employed. But by instructing the patient to breathe through the nose and to breathe heavily while the mouth is open, we obtain relaxation of the muscles of the soft palate, and in most cases, after a little training, may dispense, for diagnostic purpose, with the palate-retractor. The mirror, with its reflecting surface upward, is now passed along the tongue-depressor, until it reaches the posterior wall of the pharynx. By then raising somewhat the handle of the mirror, we obtain a view of the vomer; and by slanting the mirror first towards one side and then towards the other, the posterior nares and the orifices of the Eustachian tubes may be inspected, and the vault and posterior wall of the naso-pharynx.

The chief diseases of the larynx, grouped in accordance with their main features, may be arranged as follows:

ACUTE ORGANIC DISEASES.

Congestion, or hyperæmia.

Inflammation of the mucous membrane of the larynx—Acute laryngitis.

Œdema of the larynx.

Acute affections of the larynx and trachea as met with in children. } Catarrhal and pseudo-membranous laryngitis—False and true croup.

Specific affections—Syphilis, tuberculosis, lepra, diphtheria, erysipelas, typhoid, etc.

CHRONIC ORGANIC DISEASES.

Inflammation of the mucous membrane of a part, or of the whole—Chronic laryngitis in its various forms—Abscess.

Destruction of the cartilages.

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[1] Journal of Laryngology, 1896; Gould's Year-Book, 1897, p. 1020.

Growths and tumors of various kinds.
Ulcers, simple and specific.
Muscular degenerations, occurring after acute infectious disease (such as typhoid fever).

AFFECTIONS OF THE NERVES.

Spasm of the larynx. (Spasmodic croup and laryngismus stridulus.)
Nervous aphonia. { Functional, or purely nervous aphonia. (Hysterical, or due to debility.)
Organic, due to paralysis of the muscles of the vocal cords.
Chorea of the larynx.

Acute Laryngeal Affections.

Acute Laryngitis.—In its mild form, acute laryngitis is neither an uncommon nor a dangerous disease. In its severe form it is much more uncommon, and very much more dangerous. When it is slight, it occasions simply hoarseness; a feeling of tickling and irritation in or near the larynx; a trifling, though annoying, cough, or rather a constant disposition to clear the throat, more than a cough; and, owing in a great measure to a co-existing inflammation of the fauces, some difficulty in swallowing. The disorder passes off in the course of a few days.

When the inflammation is violent, and especially when it involves the submucous tissues, the symptoms are much aggravated. The respiration becomes seriously impeded; with each breath a wheezing or whistling noise is heard. There is but little expectoration, and the cough is distressing and painful, and has a harsh sound. The voice is hoarse, or sinks into a scarcely audible whisper; the windpipe is tender when pressed. There is in the throat a feeling of constriction, difficulty in swallowing, and fever, with a full pulse and flushed face. If the case advance unchecked, the countenance becomes distressed and pale, the lips bluish, the pulse irregular, and death sets in with all the signs of deficient aëration of the blood and of strangulation.

The disease in its graver form runs a very rapid course. If in a few days after its beginning no improvement show itself, life does not last long. Sometimes death takes place on the first day of the attack; it rarely waits for the sixth. *Œdema* of the laryngeal mucous membrane is often the consequence of the inflammation and the cause of the danger.

The marked symptoms of the perilous complaint prevent it from being overlooked, and render its discrimination easy. There is fever with dyspnœa in the *acute pulmonary affections*: but the voice remains unaltered, and they exhibit physical signs which acute laryngitis does not,—they show rales, or abnormal respiration-sounds; while in

laryngitis the murmur of the lungs is that of health, although it is sometimes enfeebled by the impediment in breathing, or obscured by the shrill sound which issues from the larynx. We find difficulty in swallowing and some hinderance in breathing in *tonsillitis;* but inspection of the oral cavity immediately detects the source of the disorder. There is difficulty in swallowing in *pharyngitis*, but there is not embarrassed breathing, or a peculiar voice, or cough, and the fauces appear dusky and injected, while they are but slightly affected in laryngitis, unless inflammation of the larynx have supervened upon that of the throat.

An affection of the larynx occurring only in winter, *laryngitis hiemalis*, has been described by Mulhall, in which the secretions form adhesive crusts, producing difficulty in speaking, or more often aphonia. This is to be diagnosticated from *laryngitis sicca*, which is a part of a general process, and follows pharyngitis sicca and atrophic rhinitis.

There is a peculiar form of inflammation of the larynx, *diffuse cellular laryngitis*, a diffuse inflammation of the cellular tissue, with lymph or pus infiltrated in the submucous tissue, to which attention has been called by Henry Gray.[1] It is a formidable affection, which bears a strong likeness to erysipelatous laryngitis, but, what is not by any means constantly the case in this disorder, the symptoms begin in the fauces and larynx; and, wholly unlike erysipelatous laryngitis, the submucous tissue is primarily attacked, and the neck becomes greatly swollen from the effused products around the larynx, trachea, and œsophagus filling its cellular tissue. The disease begins with chills, soreness of throat, and fever, soon succeeded by dyspnœa, by a dusky hue of the fauces, by enlargement of the tonsils and of the glands in the neighborhood of the jaw, and by great difficulty in swallowing. The neck increases greatly in size, the fever assumes a low type, and the patient either sinks gradually or dies asphyxiated, perishing sometimes rapidly from a speedy increase of the laryngeal tumefaction.

Other forms of inflammation of the larynx to which attention has of late years been called are *hemorrhagic laryngitis*, an acute catarrh of the larynx, attended by bleeding from the inflamed membrane, and *laryngeal rheumatism*. This generally happens in persons of rheumatic diathesis, is attended with considerable pain, and may or may not be associated with other signs of rheumatism.[2] There are cases in which laryngeal symptoms are marked, and cases without

[1] Holmes's System of Surgery, vol. iv.

[2] Archambault, Thèse de Paris, 1886.

them. Roos reports[1] several instances of rheumatic angina that terminated in attacks of general rheumatic arthritis. The principal features of rheumatic angina are excessively painful deglutition, redness and swelling of one or both tonsils; the disease is of slow development, and occurs usually without abscess-formation. It has been suggested that the joint affections are really secondary manifestations, pseudo-rheumatic in character, and that the polyarthritis belongs to the category of attenuated pyæmic infections. H. L. Wagner has found articular rheumatic affections following follicular amygdalitis, in which bacterial investigation showed that the synovial fluid obtained by tapping the joint contained the same micro-organisms as were found in the diseased tonsil.[2]

Following inflammation or ulceration of the larynx, various irregularities may occur as the result of cicatricial contraction, or adhesions between the cords, which may be studied with the aid of the laryngoscope. There are usually alterations in the voice, with attacks of dyspnœa simulating asthma, and impairment of general nutrition.

Œdema of the Larynx.—The danger in acute laryngitis of any kind is much aggravated by the precise seat of the disease. When the inflammation takes place immediately around the glottis, and causes a serous fluid to transude, *œdematous laryngitis*, the peril is greatly increased. The inspiration is audible, noisy, hissing, and labored; there is a distressing sensation of constriction or obstruction in the windpipe, and the patient makes repeated efforts, by swallowing or by hawking, to clear his throat of the substance which seems to be clogging it. His difficulty of breathing is intense, and occurs in frightful paroxysms, sometimes of a quarter of an hour's duration, in which strangulation appears to be imminent; and, indeed, often these patients do perish by strangulation.

This grave disease, *œdema of the larynx*, sometimes follows an extension of the peculiar inflammation of the throat in the exanthemata, or is of erysipelatous origin, and it occasions death quickly, and amid great suffering. But the œdema may arise without preceding acute inflammation, whether this be specific or not. It may result from long-continued pressure on the trachea or larynx, or, in exceptional instances, occur in connection with Bright's disease. Again, an effusion of serum may cause death suddenly in a person who has been laboring under a chronic laryngeal disorder. Such cases of œdema

[1] Revue de Laryngologie, etc., 1895.

[2] Rheumatic Affections of the Body due to Tonsillar Disease, Trans. Amer. Laryngol. Assoc., 1894

of the larynx are distinguished from those of active laryngeal inflammation by the absence of fever, of local tenderness, and of marked difficulty of deglutition. It is true that, if the œdematous affection ensue upon a chronic inflammation of the larynx, tenderness and an impediment in swallowing may be observed. But the history of the malady and the non-existence of fever leave little room for error.

The diagnostic sign proposed for œdema of the larynx—the swelling of the epiglottis, as ascertained by the touch—cannot be relied upon, because this swelling does not always exist to an obvious degree, and even when it does exist, is not readily determined by the finger. In the acute cases of œdematous laryngitis the laryngoscope shows a bright-red mucous membrane; sometimes the tumid epiglottis presents the appearance of two round red swellings. It is generally erect, tense, and turban-shaped. The œdema, in rare instances, may be altogether below the glottis.

Croup.—Croup is inflammation of the larynx and trachea; but it is something more. It is a spasmodic action of the muscles of the larynx, which spasmodic action gives rise to much of the peculiar cough, the stridor, and the paroxysms of dyspnœa, so characteristic of the disease. As croup is thus an affection composed, as it were, of several distinct elements, it differs somewhat according as one or the other of these elements preponderates. Thus, the inflammation may be comparatively slight, yet the spasm plays a very prominent part; or the inflammation may be very severe, and result in the formation of a false membrane. To the first class belongs the disorder known as false croup, catarrhal croup, spasmodic croup, spasmodic laryngitis; to the second, the true or membranous croup.

False or Catarrhal Croup.—This is one of the most common diseases of childhood. Its seizures happen chiefly at night; and the child that has gone to bed well, or perhaps fretful from teething, or with indigestion after a hearty supper, or with a slight catarrh, wakes up suddenly in a state of alarm, breathing with difficulty. It coughs with violence at short intervals, and the cough is loud and ringing and hoarse; and so are the voice and the cry. Each inspiration is attended with that shrill, "croupy" sound which, once heard, is never forgotten. The face is flushed, the pulse frequent, the temperature but little above the normal. The paroxysm continues in this manner for about an hour: the breathing then becomes quiet, the child falls asleep, and rests well until towards morning, when the attack is apt to be renewed. The little patient may, however, escape this altogether, and keep well; or else the paroxysm recurs the next night, or for several nights in succession. In the intervals the voice and respi-

ration are natural, there is little or no fever, little or no cough. Yet sometimes a cough occurs, during the day, which has every now and then a croupal sound; the voice, too, is slightly hoarse.

Catarrhal, or false, croup most frequently follows exposure. It is very rarely fatal. The laryngoscope shows marked congestion with swelling of the mucous membrane and copious muco-purulent secretion. Cases in which the inflammation is extensive and severe, without having led to a plastic exudation, and in which the inflammation is apt to be chiefly subglottic, approach in their persistency and in the character of their symptoms very closely to true croup. Indeed, one form of the complaint may run into the other, warranting the assumption that they are not two diseases, but only two forms of the same disease. Spasmodic croup may be a symptom of abnormalities, such as of hypertrophies or of adenoid growths in the nose or pharynx, and, if persistent, should suggest a digital or rhinoscopic examination.

The main element in the production of the symptoms of false croup is undoubtedly *spasm of the glottis*. But *laryngismus stridulus*, as laryngeal spasm or spasm of the glottis is called by many, is a neurosis which, while it may complicate any affection of the larynx and trachea, may also exist independently, from central, or direct, or reflex, causes of irritation. The laryngeal spasm may, therefore, form a distinct disorder, which differs from catarrhal croup by the absence of all inflammation and by several circumstances which proclaim its non-identity, such as its usual connection with rickets, and its frequent association with other convulsive symptoms,—with distortion of the face, rolling up of the eyes, spasmodic contraction of the hands and feet, and general convulsions. Laryngismus and tetany are often associated; indeed, by many laryngismus is looked upon as the laryngeal expression of tetany. The Trousseau sign of tetany—pressure upon the large arteries and nerves of a limb developing a paroxysm of tetany—is said to be never absent in the laryngo-spasm.[1]

Some cases of supposed purely nervous laryngeal spasm in children are undoubtedly symptomatic of laryngeal growths, or of paralysis of intrinsic muscles, and are really attacks of dyspnœa due to laryngeal obstruction. Laryngoscopic examination should be made in severe cases, even though an anæsthetic be required. Laryngismus stridulus is an affection of children under two years of age. Crying may bring on the attacks, the child dying of suffocation or during convulsions. In some cases mentioned by Mackenzie, the attack assumes the form of a sudden, almost soundless, spasm that

[1] Escherich, Address before the Tenth International Congress.

does not relax until life is extinct. Spasm of the glottis in infants may be caused by an enlarged uvula, as in cases reported by Hugel:[1] and Eustace Smith[2] cites a case of laryngeal stridor in a three months old infant, continuing since birth, in which adenoids were discovered in the naso-pharynx and removed by curetting, with complete relief.

In laryngismus, as in croup, the seizures are apt to take place at night. Generally the child has been fretful from teething, or from gastric or intestinal irritation, when suddenly an attack of difficult breathing occurs, accompanied by several loud, crowing inspirations, and by threatening suffocation; yet the paroxysm is not associated either with cough, or fever, or altered voice, or a materially changed cry. A fit of this kind may be repeated twenty or thirty times a day. It may terminate fatally in a short time; usually, however, the paroxysms are spread over weeks, or even over a longer period.

In addition to the frequent combination with other convulsive symptoms, the protracted duration of the disease, and the absence of febrile disturbance, of hoarseness, and of cough, point out the distinction between laryngeal spasm and catarrhal or spasmodic laryngitis. From bilateral palsy of the abductors of the glottis, laryngismus is readily distinguished by the great and persistent difficulty of breathing in this affection, which is a disease of adult life. Laryngeal spasm also occurs in the laryngeal crises of tabes; the absent knee-jerk and the ataxia tell us its meaning.

In the adult, glottic spasm produces symptoms to which the name of *laryngeal vertigo* has been given; the attack comes on suddenly, the patient gasps for breath and becomes unconscious and asphyxiated. In such cases there is often attendant disease of the pharynx.

True or Membranous Croup.—True croup is a formidable affection, in which there is inflammation that results in the formation of a false membrane. The plastic exudation is found lining the larynx, extending at times into the trachea or down into the bronchial tubes. With rare exceptions, cases of membranous croup are the result of infection by the Klebs-Loeffler bacillus, and are, therefore, to be regarded as laryngeal diphtheria. We shall farther on examine into this formidable affection, and determine in how far the non-diphtheritic cases can be distinguished. Let us here speak of the manifestations of ordinary membranous croup.

In the early stages of membranous croup we have the same stridulous breathing and brazen cough as in catarrhal croup. Gradually

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[1] Münchener Medicinische Wochenschrift, 1898, No. 44.

[2] Lancet, March 19, 1898.

the voice alters and becomes suppressed, and the signs of laryngeal obstruction become more evident, and shreds of membrane are expectorated.

The application of a stethoscope to the larynx or trachea does not give us much information as to the exact seat and the extent of the affection of the windpipe. Still it is not without value. It may enable us to judge of the position of the exudation, for we may occasionally hear a vibrating sound, as if a membrane were being tossed to and fro by a current of air. In a case that came under my notice some years ago, this sign was perceived with great distinctness at the lower part of the trachea and towards the commencement of the left bronchial tube; and, at the autopsy, at precisely this point was found a thick layer of membrane lying unattached in the tube. Auscultation of the lungs, by showing to what extent the air is still capable of entering them, furnishes us with a clue to the degree of the laryngeal obstruction.

Membranous croup is a disease not apt to be mistaken. When we take the symptoms collectively,—the ringing cough, the peculiar respiration, the dyspnœa aggravated in paroxysms, the changed voice, the fever, the expectoration of shreds of membrane; when we regard the comparatively short duration of the disease,—there is, with the exception of the ever-present question of diphtheritic origin, generally but one interpretation of the phenomena possible.

It is, of course, of the utmost consequence to distinguish between spasmodic laryngitis or *false croup* and membranous croup. The symptoms of the latter are far graver and more continuous, the fever is decided. But there is only one proof positive,—finding the membrane in what is coughed up or vomited up, or by a laryngoscopic examination.

The disorders, excluding diphtheria, which, next to false croup, are most likely to be mistaken for membranous croup, are: acute laryngitis, œdema of the larynx, retropharyngeal and retrolaryngeal abscesses.

Acute laryngitis in its ordinary form, such as we see in adults, is a very rare disease in children. Acute catarrhal laryngitis is in them closely connected with the phenomena of spasmodic croup; and the croupy symptoms, the changed voice, the barking cough, the paroxysmal dyspnœa, the slight or absent difficulty in swallowing, tell us what we are dealing with. In membranous croup these signs also are intensified, and we are apt to have high fever. A form of laryngitis, however, happens in children, which is very liable to be considered as croup: it is the *secondary laryngitis of the exanthemata*, especially of

variola and scarlatina. Attention to the history of the case, and to the circumstance of the inflammation having spread from the throat downward, will aid us greatly in forming a correct opinion of the disease. Yet the diagnosis is sometimes one of extreme difficulty, and examination by the microscope and culture tests will be needed to determine whether or not it is diphtheritic.

Œdema of the larynx resembles croup, in its severe or its membranous form, in the dyspnœa, the fits of suffocation and of coughing, the altered voice, and the noisy inspiration. It resembles it further in the fact that most of the symptoms do not disappear in the intervals between the paroxysms. Here is certainly a strong likeness. But the cough has not the croupal, brazen sound; expiration is comparatively unembarrassed; there is no fever, unless the œdema occur in the course of an acute affection; and, above all, œdema of the glottis is a disease of adults. Again, the history of the case often guards against error, for œdema of the larynx happens frequently, perhaps most frequently, in those who have been long laboring under chronic or ulcerative laryngitis; it is also seen among the toxic effects of iodide of potassium. In cases in which we are able to use the laryngeal mirror, the peculiar œdematous look of the parts is readily recognized.

Retropharyngeal abscesses share with croup the symptoms of dyspnœa, stridulous respiration, and altered voice. They do not share with it the peculiar cough; and, further, in croup there is not the difficulty in swallowing, or the evident tumefaction and stiffness of the neck, nor can a tumor be recognized by the touch, as it can be when an abscess is seated behind the walls of the pharynx. Moreover, the dyspnœa and the voice present somewhat different characteristics. In the case of abscess, the former is greatly augmented, or paroxysms of it are brought on, by attempts at deglutition; it is always preceded by dysphagia, is increased by pressure against the larynx, and is aggravated by the horizontal position. In croup, the patient seeks relief by throwing his head back, and, although he loses his voice and speaks in a hardly audible whisper, still the words are sufficiently distinct; while an abscess gives a nasal or gluttural tone to the voice, that often makes it impossible to understand what is being said.

Retrolaryngeal abscesses following inflammation of the areolar tissue of the retrolaryngeal space present dyspnœa, attacks of suffocation, and cough like those of croup, and run, moreover, generally an acute course; but they also present dysphagia and severe pain, occasioned by pressing on the thyroid cartilage.[1]

[1] Goix, Archives Générales de Médecine, Oct. 1882.

Abscess of the larynx bears a strong resemblance to retropharyngeal abscess, and may be, like it, mistaken for croup. Abscess of the larynx in its acute and primary form is not a frequent disease; rare in adults, it is still rarer in children. No swelling can be detected in the pharynx to account for the pain, the cough, the difficult breathing and impeded swallowing; but on close observation it is found that the larynx projects, and that there is induration at the posterior margin of the thyroid cartilage. The neck is not markedly swollen, as in diffuse inflammation of the areolar tissue. With the laryngoscope, we observe a circumscribed swelling, red at its base, and often yellowish at its apex. We do not find, as we so commonly observe in croup, that both inspiration and expiration are interfered with; the latter, indeed, may be both unembarrassed and noiseless.

Abscess of the larynx may have unsuspected causes. Poli[1] reported a case in the discharge from which the sulphur-yellow granulations of *actinomycosis* were detected. Watson Williams[2] found the Gaffky typhoid bacillus at the base of ulcers and in the structures of the larynx.

Further, croup may be mistaken for tonsilitis, for capillary bronchitis, for whooping-cough, or for the presence of foreign bodies in the larynx or trachea. But the points of distinction are evident. In *tonsillitis*, or in *tonsillar abscesses*, the breathing is not at all, or but very slightly, impaired; and a glance into the mouth is sufficient to reveal the real nature of the malady. So it is in *peritonsillar abscess*, where otherwise the suffocative attacks that are prone to happen might be misleading. In *capillary bronchitis* there is dyspnœa, as in croup; but the dyspnœa is unremitting, and associated with fine râles in the lungs, and not with a ringing cough, a harsh tracheal breathing, a hoarse voice. In *whooping-cough*, paroxysms of coughing and of obstructed respiration occur; but then follows the distinctive whoop; and there is no fever, the voice is not husky, and the child does not suffer between the spells. *Foreign bodies in the windpipe* give rise to stridulous breathing and to cough, but they do not often mimic croup closely enough to deceive; and the absence of the peculiar cough and of fever, and the history of the case, prevent error; so also does attention to the fact that the signs vary as the foreign body shifts its position. Furthermore, as Gross[3] points out, the embarrassed breathing caused by a foreign body is chiefly found in expiration.

[1] Gazzetta degli Ospitali, Naples, May 14, 1894.
[2] Journal of Laryngology and Otology, Oct. 1894.
[3] On Foreign Bodies in the Air-Passages.

The diagnosis of membranous croup has been considered connectedly, because it is convenient and practically useful to so consider it, and because I am still of the belief that there is such a disease as a membranous laryngitis which is not diphtheria, though it is rare. The strong points in the diagnosis of non-diphtheritic membranous croup are: the gradual origin and the slow deepening of the symptoms; the fact that no membranes appear in other localities; that the disease has a laryngeal onset,—though this may happen also in diphtheria,—and, above all, the absence of the Klebs-Loeffler bacillus in any shreds of membrane in the expectoration. In discussing laryngeal diphtheria the matter is further examined into.

Chronic Laryngeal Affections.

Of the chronic diseases of the larynx, chronic inflammation of the mucous membrane and thickening and ulceration are the most common.

Chronic Laryngitis.—Alteration of the voice, cough, and an uneasy feeling in the larynx are the main symptoms. The cough is at first dry, but when of any standing is followed by a yellowish opaque expectoration. It either presents nothing peculiar in its tone, or else is harsh and barking. The breathing is little, if at all, embarrassed, except when the mucous textures are greatly thickened or ulcerated. In that case there is dyspnœa, the respiration is apt to be noisy and the voice completely lost, because the vocal cords have also suffered. There is, moreover, considerable pain on pressure; the sputum is muco-purulent, or else purulent and streaked with blood; and sometimes, if the cartilages also be involved, fragments of them are expectorated, and by the touch we recognize the changed state of the tube.

The symptoms of chronic laryngitis are mostly not purely local. Chronic laryngitis is frequently, indeed, found to be connected with a broken constitution, because the inflammation of the larynx, both in its simple and in its ulcerated form, is often combined with tuberculosis, or with syphilis. In every patient, therefore, suffering from chronic laryngitis, we must endeavor to ascertain whether either of these morbid conditions is present. Chronic laryngitis frequently turns out, on thorough examination, to be laryngitis linked to a serious pulmonary difficulty; or we detect ulcers in the pharynx associated with those in the larynx and cicatrices, and are enabled to trace clearly the ravages of constitutional syphilis.

As seen with the laryngoscope in chronic laryngitis, hyperæmia, general or partial, is present, associated in cases of long standing with

considerable and uniform swelling of the mucous membrane; the vocal cords are often uneven at their edges, and there may be, chiefly between the arytenoid cartilages, superficial ulcers. Papillary growths upon the edges of the vocal bands may follow inflammation or repeated attacks of hyperæmia.

Chronic laryngitis is liable to be mistaken for an aneurism of the aorta, or, more strictly speaking, an aneurism of the aorta is liable to be regarded and treated as a case of chronic laryngitis. The distinction, as will hereafter be shown, is mainly made by attention to the physical signs; often the paralysis of a vocal cord is of great significance.

Cases of functional or *nervous aphonia*, too, are sometimes confounded with chronic laryngitis; and it is by no means always easy to avoid this error. The loss of voice may be either partial or complete. It not infrequently comes on without any previous warning; and this fact aids us greatly in diagnosis. So does the absence of cough, of expectoration, of local pain, and of all difficulty in breathing; for none of these symptoms are commonly observed in aphonia which is solely nervous. One of the causes of the disorder is overstimulation of the vocal nerves, by straining the voice in singing or in speaking. We also meet with it occasioned by narcotics or by lead poisoning, and perhaps most frequently as a reflex manifestation, due to irritation of the intestines by worms, or to a disorder of the uterine system. In these instances of nervous aphonia the voice suddenly disappears and as suddenly reappears, a phenomenon not unusual in the aphonia of hysteria; and we may have from impaired, but not wholly lost, power the voice absent only for some hours daily. It is evident that in all cases of nervous aphonia the laryngoscope will assist us greatly; it shows the true condition of the parts, as regards both their structure and their mobility. It also aids us in distinguishing these laryngeal disorders from cases of *aphonia* due to want of strength in breathing,—to want of power in expiration.

Enlarged bronchial and cervical glands, or an aneurism which compresses the laryngeal nerves, also produce hoarseness, and ultimately complete loss of voice. Under such circumstances there is a short cough, attended often with loud tracheal râles; and we observe attacks of dyspnœa, with a noisy, hissing respiration. The practical lesson which all such cases teach, is to remember that the symptom considered most characteristic of chronic laryngeal inflammation—the altered voice—may occur when no laryngitis exists: also to examine with the laryngoscope, and to note the effect of palsy of the muscles, the result of nerve-pressure. In thoracic aneurism, pressure symp-

toms—such as dyspnœa and altered voice, with paralysis of laryngeal muscles—may be produced either by pressure upon the recurrent laryngeal nerve, which on the left side passes around the arch of the aorta, or upon the vagus. Pressure upon the vagus will give rise to abductor paralysis of the corresponding side, with adductor spasm of the laryngeal muscles of the opposite side, the spasmodic movements being intermittent. Pressure upon the one recurrent nerve causes one-sided abductor paralysis, the degree of pressure determining the amount of paralysis; thus, when complete, there is entire loss of voice, when incomplete the voice may be hoarse, whispering, or unimpaired. This condition of one-sided abductor paralysis may be caused by pressure from an enlarged cervical gland, by aneurism of the arch of the aorta, and by various forms of mediastinal tumors. Pressure upon one vagus, inducing double adductor spasm, produces serious dyspnœa and difficult phonation; but pressure on one of the recurrent nerves may occasion intermittent dyspnœa that is usually not troublesome, and scarcely affects phonation. Major's researches have given us much of this definite knowledge.

Now, in the nervous forms of aphonia just mentioned, with the exception of those caused by pressure, the loss of voice is due to deficient power, and the cords move sluggishly or not at all. When the disorder reaches a high degree, we perceive, on looking into the laryngeal mirror, that the vocal cords do not approximate as the patient attempts to say *a* or *o*. But, besides these cases, owing to general want of force, we find cases of *spasm of the tensors of the vocal cords* with most peculiar, partially interrupted voice; and of absolute *paralysis of individual muscles*, as of one adductor of a cord; or of one or both posterior crico-arytenoids, or abductors; or of the crico-thyroids, or tensors. In some of these there is considerable dyspnœa, with noisy breathing; in all the laryngoscope affords the only means of diagnosis. In paralysis of the external tensors of the vocal cords, the crico-thyroid muscles, there is inability to use the higher notes with freedom; the voice is rough or entirely lost, and viewed with the mirror we find a wavy outline of the glottis, convexity of the upper surface of vocal bands on expiration and phonation, and slight concavity on forcible inspiration. The contraction of the muscles, which in the healthy subject can be felt externally during phonation, is completely absent. This form of disorder most frequently results from overstraining the voice; it may be caused by cold,[1] and is apt to be bilateral. Palsy of the thyro-epiglottic muscles has its usual origin

[1] Major, Proceed. Amer. Laryng. Assoc., 1892, p. 10.

in diphtheria. The epiglottis stands erect, and does not move during attempts at deglutition. In palsy of the relaxors of the vocal cords, the thyro-arytenoid muscles, the deep tones are nearly gone. It is often unilateral, and comes mostly from overexertion of the voice during catarrhal laryngitis. Viewed in the laryngeal mirror, the edges of the cords do not approach in the median line, and they seem excavated. In paralysis of the posterior crico-arytenoid muscles, the glottis is seen as a narrow slit, becoming still narrower during inspiration. There is no disturbance of voice, and scarcely any sign of laryngeal catarrh, but there is marked and noisy laryngeal dyspnœa. This paralysis of the abductors may happen from compression of the recurrent nerves by an organic stricture of the œsophagus.[1] Alex. W. MacCoy has reported three cases of bilateral abductor paralysis during or after typhoid fever, which he attributed to degeneration, the result of the fever process, in the posterior crico-arytenoid muscles.[2]

Bilateral paralysis of the adductors is a common disorder occurring in connection with locomotor ataxia and affections of the brain and of the medulla. Paralysis of the muscles of the larynx occurring in typhoid fever has been observed by Mendel and Bonlay[3] and Ludwik Przedborski.[4] It happens both during the fever and in convalescence. Nearly all of the muscles of the larynx may suffer in this way; the paralysis appears first in the constrictors of the glottis, and spreads to the remaining adductors; finally the abductors are affected, and ultimately a total palsy of the recurrent laryngeal nerve is the result. Recovery of function may follow in from one to three weeks. There is a tendency for the affection to become chronic, yet the prognosis is usually favorable. When the abductors of the larynx and the posterior crico-arytenoids are both paralyzed, the vocal cords remain near the median line, and do not separate during the act of inspiration; such cases are liable to perish from suffocation during an attack of dyspnœa. Thomas[5] has reported a case of paralysis of both recurrent laryngeal nerves consecutive to typhoid fever, which he found to be due to diffuse neuritis. Unilateral paralysis of the adductors is more rare; it accompanies malignant disease of the œsophagus, aneurism of the aorta, and, exceptionally, metallic poison-

[1] Case of Dujardin, Annales des Maladies de l'Oreille, 1887.

[2] Section on Otology and Laryngology, College of Physicians of Philadelphia, Philadelphia Medical Journal, 1899.

[3] Archives Gen. de Med.; Revue de Laryngologie, 1895.

[4] Klin. Vortrage, N. F., No. 182, May, 1897.

[5] Revue de Laryngologie, 1898, No. 20.

ing, as lead and arsenic. It sometimes follows exposure to cold, or attends rheumatism or phthisis. When met with in connection with paralysis of the same side of palate or tongue, it is centric, at times bulbar. E. Fletcher Ingals has described cases thought to be hysterical in origin. We also encounter *sensory neuroses* of the larynx, and among these hyperæsthesia is common.

Chronic laryngitis, or rather its chief symptom, loss of voice, is at times *feigned;* and the deception may be kept up for an indefinite period. Yet we possess, in the use of anæsthetics, the means of detecting the fraud at any moment. Just before the impostor falls into the deep sleep produced by ether, or as he is recovering from the insensibility it occasions, his will no longer controls his voice, and he speaks in his natural tone, or even screams violently.

Now, under the term chronic laryngitis, which formerly, for want of more precise knowledge, was made to embrace most kinds of chronic diseases of the larynx, many different morbid processes are embraced, the exact nature and seat of which we may discriminate by the laryngoscope. Thus, the disorder may be wholly, or almost wholly, confined to the *epiglottis.* We may find this structure highly congested and enlarged; we may be able to note that it is pendent, nearly completely covering the glottis; and it is frequently the seat of ulceration. The attending symptoms in any case are those regarded as characteristic of a greater or less degree of laryngeal inflammation. In instances of ulceration there is soreness with pain in swallowing, hoarseness and irritative cough, followed at times by blood-streaked expectoration. The ulceration may terminate in total destruction of the epiglottis. A turban-shaped swollen epiglottis is often met with in phthisis associated with pyriform swelling of the arytenoids. Pallor of these structures, indeed of the whole larynx, is one of the early signs of pulmonary tuberculosis, as Cohen has pointed out.

When the *vocal cords* are affected, we recognize in the laryngeal mirror either their reddening in part or entirely, or their induration and thickening, or we observe œdematous swelling in and around them, or their ulceration; and we can usually detect during breathing and phonation their impaired action. The inflammatory redness may be only in one cord. Small collections of mucus are often found adhering to different parts of the laryngeal membrane. Now, all these conditions are generally combined with marked aphonia; the voice, indeed, may be reduced to the merest whisper. Venous congestion of the larynx is so rare an affection that Mackenzie has met with but four cases of

it.[1] In making our diagnosis we must always be careful to find out if the laryngeal phenomena be not secondary, forming part of a general morbid state, such as dropsy, tuberculosis, syphilis, or changes in the blood. Chronic hypertrophy of the ventricular bands is the result of inflammatory thickening, and, as Tauber[2] proves, occurs mostly in those who use the voice much in their professional vocations. Türck has given the name of "chorditis tuberosa" to a condition of the vocal bands in singers, in which are found in the upper plane of the bands a peculiar uneven surface and white opaque spots as large as poppy-seeds.[3] It has been suggested that capillary fibroma or even malignant disease may have an inflammatory origin.

Paralysis of one vocal cord may exist, with immobility of one side of the larynx, and yet voice may be preserved; the healthy cord, as in cases narrated by Bosworth, swinging over to the paralyzed side, so as to make up for the loss of power on that side. Voice may even exist, to a restricted extent, not only without vocal cords, but after entire extirpation of the larynx, as in the remarkable case reported by J. Solis Cohen,[4] in which the larynx was removed for malignant growth, and the trachea permanently fixed in the neck. After gulping some air into the gullet and throat, the patient was able to talk, and even to sing, by skilfully using his pharyngeal muscles.

Alteration of the voice, mumbling speech, as though there were some difficulty in closing the glottis, while the movements of the vocal cords appear normal as seen with the laryngoscope, without true aphasia, is mentioned by John N. Mackenzie as a symptom in a case of bulbar disease.[5]

Diseases of the cartilages and of the perichondrium are most frequently encountered in connection with tuberculosis, syphilis, and typhoid fever. The affection often begins in the submucous tissue, and the ulceration spreads until the cartilaginous parts of the larynx are involved. The arytenoid cartilages are generally first attacked; and portions of these cartilages may be thrown off and expelled. At times pus is formed which gives rise to swellings that can be recognized by the aid of the laryngeal mirror; sometimes a displacement of the cartilages takes place, before any portion of them is completely separated, and the most distressing and dangerous attacks of suffocation result; or the perichondritis may lead to the development of

[1] Diseases of the Throat and Nose, vol. i., 1880.

[2] Cincinnati Lancet, 1887.

[3] Klinik der Krankheiten des Kehlkopfes, Wein, 1866.

[4] Pharyngeal Voice, Transactions of the Amer. Laryng. Assoc., 1894.

[5] Transactions Amer. Laryng. Assoc., New York, 1891, p. 6.

bone-substance and a constriction of the tube. In some instances the purulent collection presses on a vocal cord, which, with the laryngoscope, may seem to be immovable.

This instrument reveals very generally the ravages the disease has committed; and we are thus enabled to form an opinion as to how far the destruction has progressed, and which of the soft parts as well as of the cartilages are involved. Leaving out the frequent perichondritis and caries of the cartilages which follow the deposition of *tubercle*, we find in laryngeal phthisis considerable swelling and ulceration of the epiglottis, and often semisolid pyriform swellings of the ary-epiglottic folds. The thickening is more regular and uniform than that of syphilis, and the tubercular ulcers not large and solid as in this affection, but small and numerous, and both vocal cords are involved; while in this as in every other respect syphilis is more apt to be local and unilateral. Tubercle bacilli are found in the discharge from the laryngeal ulcer, and in catarrhal ulceration the ulcers are generally very superficial and on the vocal cords. The symptoms of laryngeal phthisis are difficulty in breathing and in swallowing, local pain and soreness, a greatly altered or a lost voice, and a distressing, harsh cough, which is followed at times by purulent expectoration. Besides, we find the manifestations of disease of the lungs. But it occasionally happens that we encounter cases of tuberculous ulcers with abundant bacilli, in which no lung disease exists; and it is not uncommon to find the tubercular disease of the larynx preceding that of the lungs. At times we note syphilitic and tubercular ulcers in combination. We may also meet with catarrhal ulcers where there is tubercular disease of the lungs. A means of diagnosticating *syphilitic affections* of the larynx from others has been proposed and practised by Justus.[1] It is based upon the fact that after the use of mercury by inunction or by hypodermic injection in a patient affected by syphilis, a sharp fall in the percentage of hæmoglobin occurs, within the few hours immediately following the introduction of the remedy into the system. Later, the proportion of hæmoglobin increases to a point above where it was before. Justus observed the sudden fall of ten to twenty per cent. in the hæmoglobin, following this use of mercury, in over three hundred cases of syphilis. No effect of the kind was observed when the mercury was administered by the mouth. This has been called Justus's test, and has been found applicable to cases of ulceration of the larynx, in which there was a doubt as to the character of the disease.

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[1] Virchow's Archiv, vol. cxl., also cxlviii., 1897.

The diagnosis between *pachydermia of the larynx* and the interarytenoid tumor of phthisis is that in the latter the swelling is distinctly a tumor, with more or less well-defined margin. The color is usually red or pink; in pachydermia it is whitish-gray or only slightly pink.[1] Ulceration occurs in pachydermia only exceptionally and as a complication; it is common in phthisis of the larynx.

As the result of disease of the cartilage and of the perichondrium, especially as the result of the process of cicatrization, we may have stricture of the larynx and trachea; for this is, in truth, the most common origin of *laryngeal stenosis*. The inspiration is prolonged and noisy; the voice is generally, although not of necessity, affected. There is dyspnœa, and with the laryngoscope we can see how greatly the caliber of the tube has been encroached upon. Cicatrization is common after syphilis, but Cohen's case[2] proves that it may occur spontaneously also in tubercular ulcerations. Adhesions may be congenital, a web-like membrane uniting the vocal cords through a part of their extent, as in a case of Morell Mackenzie's. According to Paltauf,[3] primary stenosis of the larynx may be caused by scleroma, which may develop early in the larynx. The diagnosis depends upon the detection of the characteristic minute structures.

Ulcers in the posterior walls of the larynx give rise, as a rule, to distressing cough. *Tumors* of the larynx and polypoid growths in its interior have as their symptoms cough, altered voice, a steadily increasing difficulty in breathing, and attacks of suffocation for which nothing in the lungs or heart or great vessels accounts. But the laryngoscope alone tells us the true meaning of these symptoms.

New growths may occur in the larynx, of the benign form. Papilloma, papillary fibroma, is probably the most common; myxoma is rare; fibromyxoma and fibroma unusual. Malignant disease in various forms may affect the structures of the larynx. A positive diagnosis can be made only with the aid of the microscope. Yet the detection, at the seat of the larynx, of a growing tumor, accompanied by severe cough, by sanious sputum, by signs of destruction of tissue, by perichondritis and exfoliation of the laryngeal cartilages, by hemorrhages, and by emaciation, warrants the diagnosis of *cancer*, whether or not much pain be present. This may be confirmed by the subsequent rapid development of the malignant disease, associated with a musty odor of the breath, distress in swallowing, bloody expectora-

[1] McBride, Edinburgh Medical Journal, April, 1893.

[2] Amer. Journ. Med. Sci., Dec. 1888.

[3] Sajous, Annual of Univ. Med. Sci., 1893.

tion, and cachexia. In some instances gangrenous pneumonia occurs. *Polypi* in the larynx may sometimes be seen by depressing and dragging forward the tongue until the epiglottis is brought into view. But as regards polypi, or, indeed, any form of morbid growth, we possess in the laryngoscope the only certain means of detecting them. These laryngeal growths vary much in size and in color; they are often seated at the anterior free edges of the true cords, or still more generally just above or just below their origin, and are, as a rule, readily discerned. Sometimes they may exist for years, merely producing changes in the voice and some cough, but no very great distress; or they may lead to fits of strangulation and to sudden death. It is impossible to be sure of their nature without repeatedly examining portions of them. *Papillomas* are usually cauliflower-like or in bunches; they occupy most frequently the vocal cords, while *sarcomas* are oftenest found at the anterior portion of the larynx. *Cysts* of the vocal cords are much rarer than other forms of growths; they sometimes rupture spontaneously, and the hoarse voice quickly clears.[1] Myxomata of the larynx and the epiglottis, according to Van der Poel,[2] may be manifestations of pernicious anæmia. They differ from cysts in being a pure, gelatinous growth characterized by stellate fusiform cells embedded in a homogeneous, or finely fibrillated, soft, basement substance. Many cases that are classed as cysts would come under the head of myxoma if the aid of the microscope had been sought.

Before concluding these remarks on diseases of the larynx, it may be thought necessary to point out the differences between them and diseases of the *trachea*. But affections of the trachea need not be separately considered. Lying between the larynx and the bronchi, the trachea commonly shares in their disorders. Thus, we have seen croup to be a malady in which both larynx and trachea are involved. Slight inflammation of the trachea occurs constantly in slight attacks of laryngitis or of bronchitis. *Ulcers* in the trachea may exist without ulceration of the larynx; but then they usually escape detection. Sometimes, however, they reveal themselves by a constant pain at the lower portion of the neck and the upper part of the sternum, joined to all the symptoms of ulceration of the larynx except the impaired voice. *Morbid growths*, too, occur in the trachea, —cancer, carcinoma, syphilitic growths,—as they do in the larynx,

[1] Heinze, Archives of Laryngology, New York, 1880.

[2] American Laryngological Association, 1890.

and the tube may be altered in form and in structure. Vegetations also form in the trachea after tracheotomy.[1] We can make use of the laryngoscope to assist us in the diagnosis of any of the forms of tracheal disease referred to. Yet the instrument is not always available; for it is only under favorable circumstances that the entire extent of the trachea can be seen.

In *narrowing* of the trachea the bronchial tubes are also at the same time often narrowed. The stenosis may be caused by external compression, as from a goitre, from an aneurism, or from a mediastinal tumor; or the constriction may be due to some cause, such as new formations, in the walls of the tubes. The chief symptoms are the same in either case; and they are, long-drawn-out respiratory acts, noisy breathing, especially in paroxysms, dyspnœa, particularly marked in inspiration, epigastric retraction, feebleness or absence of vesicular murmur, with clear pulmonary resonance, loud wheezing heard with the stethoscope at or near the place of constriction, and voice slightly, if at all, impaired. This, the normal appearance of the larynx as shown by the laryngoscope, and the almost imperceptible motion of the windpipe during breathing,[2] are of great value in distinguishing a tracheal stenosis from a laryngeal affection. A *bronchial stenosis* is chiefly discriminated by the signs of the constriction being one-sided, and attended with marked thrill of the thoracic wall of the affected side, and with loud sounds issuing from it, loud enough to be heard at a distance. Subglottic œdema may be detected by the laryngoscope on deep inspiration. Over the trachea, the tracheal breathing may have become inaudible in stenosis of both main bronchial tubes.[3]

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[1] See cases collected by Petel, Des Polypes de la Trachée, Paris, 1879.

[2] Gerhardt; also Riegel, in Ziemssen's Cyclopædia.

[3] Aufrecht, Deut. Arch. f. klin. Med., lviii. 4 and 5, 1897, p. 484.

CHAPTER IV.

DISEASES OF THE CHEST.

An examination of the diseases of the chest must be prefaced by a description of the methods of investigation which have given to their diagnosis such certainty. The same methods may be applied in the study of the maladies of other parts of the body, but they are of special service in the recognition of thoracic disorders, and will be here, therefore, most appropriately considered.

The discrimination of disease by the eye, the ear, the touch, in fact, by the direct aid of the senses, is called *physical diagnosis;* the signs thus ascertained are connected with perceptible alterations in the material properties or physical nature of structures,—such as alterations in their form, their density, or their sounds,—and are known as *physical signs.*

Physical signs are, then, the exponents of physical conditions, and of nothing more. But as the same physical conditions may occur in various diseases, so may the same physical signs occur in various diseases. An isolated sign is, therefore, not diagnostic of any particular malady. It reveals usually an anatomical change; but it does not determine the disorder occasioning this change. The subject may be much simplified by laying less stress on individual signs, and by grouping them together according as their association becomes distinctive of certain well-marked physical states. Morbid anatomy tells us in what diseases these states are commonly found. It is in conformity with these views that I shall attempt to delineate the signs of thoracic affections.

For the sake of convenience, the surface of the chest has been mapped out into regions. Various arrangements of these have been made by different authors. The simplest division of the chest is into anterior, posterior, and lateral surfaces. The regions into which the anterior surface may be, for practical uses, subdivided, are an upper region, extending from just above the clavicle to the fourth rib, and a lower region, from the fourth rib downward. Posteriorly, also, there are an upper and a lower part of the chest to be specially examined. It is hardly necessary to say that all these regions are double,—the

same on each side of the chest. Many more divisions are usually made; but they are perplexing to the student, and of doubtful value. The artificial boundaries generally laid down are, indeed, too minute, and yet not minute enough; they are too minute for ordinary purposes, not minute enough when it is desirable to localize a physical sign. Whenever this is requisite, instead of resorting to the names of the regions usually employed, I think it preferable to designate the seat of the sign with reference to some fixed anatomical point. This may be done for the anterior part of the chest by indicating the distance above or below the clavicle, or near what part of the sternum, or at which rib, or spreading over how many intercostal spaces, the sign in question is perceived. At the posterior part of the chest, the spinous ridge of the scapula, its lower angle, and the spinal column, serve as landmarks. For most clinical purposes, it is only needed to study the region above the spinous process of the scapula, as separate from the space below. But in some instances it may be necessary to notice the region between the scapulæ, interscapular, or that extending from the lower angle of the bone to the limits of the chest, infrascapular.

Let us now examine the different methods of physical diagnosis, particularly in their relation to pulmonary diseases.

SECTION I.

DISEASES OF THE LUNGS.

The Different Methods of Physical Diagnosis, and the Physical Signs of Pulmonary Diseases.

INSPECTION.

If the chest be examined with the eye, we obtain an idea of its form, size, and movements. In health this inspection shows us that the two sides of the chest are, to a great extent, symmetrical in form, as well as in size and in movement. Both sides rise equally during inspiration and sink equally during expiration. On both sides the motion of inspiration is longer than that of expiration, and the pause between them extremely slight.

This *respiratory movement* is visible over the whole thorax. In males it is most distinct at the lower portions of the chest; in females it is most perceptible at the upper. In healthy adults the lungs expand from sixteen to twenty times in a minute. In certain pulmonary

affections, especially in pneumonia, the number of respirations often exceeds fifty in a minute. But hurried breathing and changed movements of the thorax occur independently of diseases of the lung, as in an hysterical paroxysm. Where the diaphragm does not descend, as in consequence of peritonitis or of abdominal dropsy or of tumors, the breathing is rapid, and is perceptible at the upper parts of the chest. Again, the thoracic movements may be distinct on one side and hardly noticeable on the other, as in pleurisy or in pneumothorax. Lastly, as happens in some cerebral lesions, the motions of the chest may be very slow and labored, or irregular, or they may have apparently ceased, and the breathing be altogether abdominal.

The *form* of the chest is sometimes strikingly altered. Congenital malformations and curvatures of the spine modify it; so do intrathoracic affections. Frequently the chest presents a retracted or an expanded look. Retraction denotes diminished size of the lung, and, if one-sided, is usually indicative either of chronic changes in the lung-tissue, as in chronic pneumonia or in tubercular lungs, or of false membranes which bind down the lung; or it is found in a very marked manner in empyema with external opening. Expansion of the chest is met with in emphysema, in pneumothorax, and in pleuritic effusion. A local or partial expansion, or bulging, may be encountered in the latter disease, or it may depend on thoracic tumors, on pericardial effusions, or on hypertrophy of the heart.

A mode of inspection of value in certain cases is the *diaphragm phenomenon* to which Litten[1] has called attention. In a person lying with his feet pointing towards a window, there can be seen during deep breathing a shadow from about the seventh to the ninth rib; it flits down during inspiration, it ascends during expiration. This shadow is nearly or wholly absent when fluid or air occupies the pleural cavity; also in obliteration of the cavity by adhesions; in intrathoracic tumors at the lower part of the chest; in pneumonias of the lower lobe, and in extensive emphysema of the lungs. Tumors under the diaphragm or accumulations of fluid in the abdomen do not impair the sign, unless they are very large, nor do enlargements of the liver or spleen. The shadow phenomenon becomes thus of much value in distinguishing morbid states above from those below the diaphragm. Litten holds that when the excursion of the diaphragm during forced breathing is less than two and a half inches the condition is abnormal. In very fat persons the shadow cannot generally be seen; muscular weakness very decidedly limits it. It is also much limited in phthisis, as both

[1] Deutsches Medicinische Wochenschrift, 1892.

Rumpf[1] and Cabot[2] prove, but its diminution may show only on the affected side.

A new and most valuable means of inspection has been discovered in the *Röntgen light*. These X-ray examinations have solved the problem of looking under the skin and making deep-seated parts visible, and of giving us photographs for permanent study, while by the adaptation of a simple instrument, the fluoroscope, or by the fluoroscope screen, we can do so more quickly and see the parts in motion. There are immense possibilities in this new mode of inspection; and while, thus far, it has proved itself of the greatest use to surgery in detecting, for instance, changes in the bones, fractures, dislocations, and foreign bodies, it has also shown its value in medicine. Among its contributions to this we may note the information it gives us concerning rickets, gouty deposits about the joints and under the skin, rheumatoid arthritis, the presence of renal calculi. Very valuable is the added insight gained by X-ray examinations in diseases of the lungs and heart, especially in giving us accurate information as to the size and movements of the latter. In the recognition, too, of arteriosclerosis and of thoracic as well as abdominal aneurism, the rays have proved themselves of the greatest use, particularly as they have enabled us to detect them in the early stages.

As regards the lungs, we have gained much precise topographical as well as pathological knowledge.[3] The exact relations of the bronchial tree to both the posterior and anterior thoracic walls have been clearly ascertained by skiagraphy.[4] On the posterior wall in the adult the course of the left bronchus is found to be from a point to the right of the fourth thoracic spine to a point on the eighth rib three inches to the left of the spine; the course of the right bronchus to a point on the eighth rib two inches to the right of the spine. With reference to the anterior wall of the chest, the point of bifurcation in the adult is just internal to the junction of the lower border of the second costal cartilage with the sternum; in children it is opposite the third chondro-sternal articulation.

The fluoroscope is better for most examinations of the lungs than the X-ray photograph, or skiagraph; it is much quicker, and shows us the parts in motion. The patient is best examined standing up.

[1] Berliner klinische Wochenschrift, No. vi., 1897.

[2] Medical News, April, 1899.

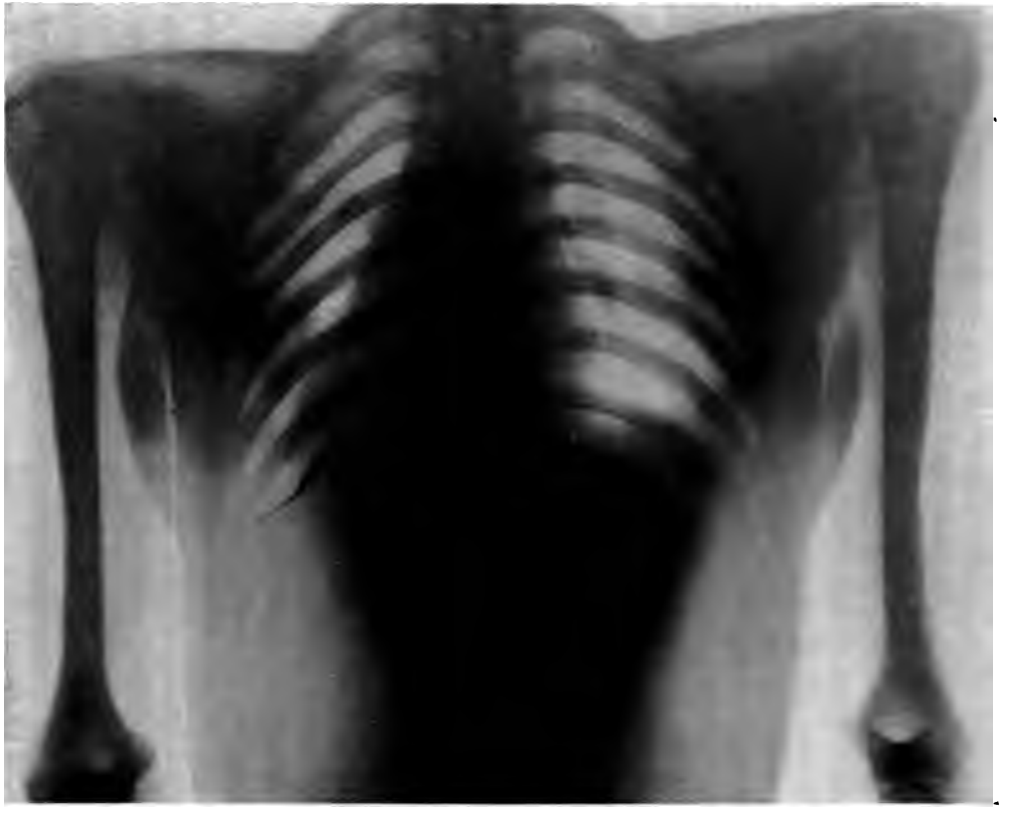
[3] See cases published in several valuable contributions by F. H. Williams, Medical News, vol. lxxii.; the American Journal of the Medical Sciences, June, 1899; also the Transactions of the Association of American Physicians, 1897.

[4] Blake, Amer. Journ. Med. Sci., March, 1899.

PLATE I.

Skiagraph in the case of a girl, sixteen years of age, at the Pennsylvania Hospital. The picture shows distinctly the outlines of a normal thorax, and the relations of the heart to the chest walls. The heart is very slightly enlarged. The case was one of mitral disease.

PLATE I.



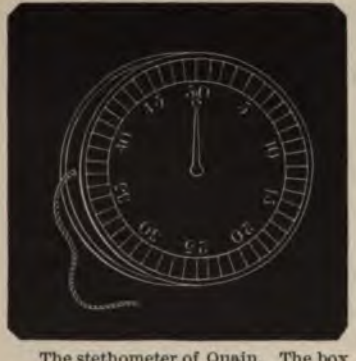
Skiagraph in the case of a girl, sixteen years of age, at the Pennsylvania Hospital. The picture shows distinctly

MENSURATION.

To measure the circumference of the chest or of the abdomen, or to ascertain the distance from one portion of the surface to the other, a graduated tape is all that is required. To attain the former object, the spinous process of a vertebra is chosen as a fixed point, and the tape is thence passed round the body to the median line, first on one side, then on the other, taking care that it be applied evenly to the skin, and that the level of the measurement be the same on both sides. If we wish to obtain the longitudinal diameter, the line from the clavicle to the base of the chest is taken. Where the chest is deformed, a chain with links may be used in place of the tape.

In estimating the size of the chest in disease, it must be borne in mind that even in health its two sides vary widely. The half-circle on the right side is, in right-handed persons, at least half an inch larger than the half-circle on the left. But the measurements, to be trusted, must be performed while the patient is holding his breath in expiration. In inspiration the girth of the chest is increased from two to three inches. In well-developed men it measures at the upper part, at the level of the nipples, about thirty-three to thirty-four inches during expiration. Otis,[1] as the result of one thousand measurements, gives the average girth in men as 34 inches in repose, and 36.1 inches inflated; and in well-developed women as 29.5 in repose and 31.5 inflated; while the depth of chest in repose in men is set down at 7.5, and in women at 6.9 inches.

FIG. 18.



The stethometer of Quain. The box is placed on the sternum, and the string carried around the chest. One revolution of the index, which is moved by a rack attached to the string, indicates an inch of motion in the chest.

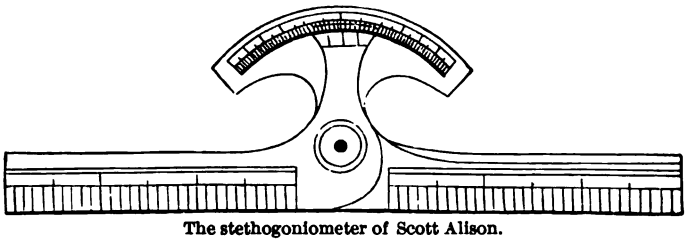
If it be desirable to ascertain in how far the respiratory acts modify the dimensions of the chest or of the abdomen, this may be readily effected by the ingenious "chest-measurer" of Sibson, or by the "stethometer" of Quain or of Carroll, or by the instrument of Démény; or the respiratory curves can be traced and studied by the atmograph of Burdon Sanderson, or by the pneumograph.

The transverse diameter—the breadth—of the chest may be determined by means of a pair of calipers, arranged specially for the pur-

[1] Boston Medical and Surgical Journal, April, 1895.

pose; and the curves or flatness of the surface may be ascertained, should it be necessary, by Alison's stethogoniometer (Fig. 19); but it is rarely necessary. In fact, these minute measurements, however interesting to the physiologist, have not much clinical value.

FIG. 19.



The stethogoniometer of Scott Alison.

Mensuration may be employed not only to judge of the size of the chest and of its movements, but also to ascertain the amount of air which is received into the lungs. The instrument used for this object is the *spirometer*. But the results obtained are not on the whole valuable. Sex, weight, age, and height have to be taken into account. For every inch above five feet, eight cubic inches are to be added to the healthy standard; for the height of five feet, the breathing volume is one hundred and seventy-four cubic inches. Otis[1] estimates the average lung capacity for height in males between sixteen and forty years of age at twenty-three cubic centimetres for every centimetre of height; and in women about nineteen years of age at fifteen cubic centimetres for each centimetre of height. Brehmer makes it between sixteen and seventeen and a half cubic centimetres. The vital capacity may be increased by practice, with the spirometer, or by the use of pneumatic instruments designed to breathe in compressed air or to breathe out into rarefied air.

Waldenburg measures the force in respiration by a special apparatus, and has introduced *pneumatometry* as a means of diagnosis. In health the power exerted in expiration is greater than in inspiration by from twenty to thirty millimetres. In some affections the expiratory pressure is largely diminished, as in emphysema and asthma, while in the forms of phthisis the force of inspiration is much lessened.

PALPATION.

Palpation, or the application of the hand, confirms the results obtained by inspection and mensuration as to size, form, and movements. It may, in addition, be employed to determine spots of sore-

[1] Loc. cit.

ness, the density and condition of tumors, the state of the thoracic walls, the frequency of the breathing, and the action of the heart. The hand may further be of service as a means of distinguishing vibrations produced by rhonchi, *rhonchal fremitus*, or by the voice, *vocal fremitus;* or it may detect fluid by the sense of fluctuation it imparts, or a roughened serous membrane by the friction fremitus. When both fluid and air are present in a large hollow space, by shaking the patient a distinct vibration of the parietes is felt, accompanied by a splashing sound, known as the Hippocratic or succussion sound.

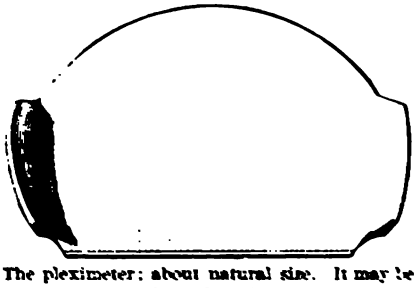
Palpation is to be practised by applying the palmar surface of one or of several fingers evenly, and without too much pressure, on the part to be examined.

PERCUSSION.

By percussing or striking bodies we elicit sounds by which we judge of their composition. Percussion was first practised by striking directly with the hand over the organs to be explored; a method which has given way to mediate percussion. The media used to receive the blow are various: a disk or plate of ivory, or of leather; a piece of india-rubber; or the middle finger of the left hand. The finger answers best for percussion of the chest.

When the finger is employed, it ought to be applied with its palmar surface firmly pressed against the chest, and as nearly parallel as possible to the ribs. One or two fingers of the other hand may then be used to tap with,—for the finger is, for ordinary purposes, better than any of the percussion hammers,—the greatest attention being paid to the circumstance that the percussing finger strikes perpendicularly, whatever plexímeter be used, and not slantingly, as is too generally the case. The whole movement should proceed from the wrist, and only from the wrist, and ought not to be too rapid, or unequal, or of great force. No fault is so often committed as that of raising the finger used as a plexímeter from the surface,—thus obtaining the sound of the finger, and not that of the organ to be percussed,—unless it be the fault of striking with great force. Forcible percussion is of use only when the sound of deep-seated organs is to be brought out.

Fig. 20.



The plexímeter: about natural size. It may be conveniently made of hard rubber.

The main sounds elicited by percussion may be designated as dull, clear, and tympanitic. Of course, these, like all other sounds, may differ in strength, in duration, and in pitch.

A *dull* sound denotes absence of air. It is the sound both of fluids and of solids. It is, thus, the sound sent forth from the airless viscera,—from the liver, spleen, and heart. When it takes the place of the pulmonary sound, it bespeaks consolidation, from whatever cause induced, or the presence of something which checks the normal vibrations of the lung-texture. Dulness is always associated with an increased sense of resistance to the percussing finger, and over parts emitting it the vibrations of the tuning-fork, which Bass has introduced into diagnosis, are weak, while they are loud over normal pulmonary structure.

A *clear* sound is produced by a series of marked and unhindered vibrations which are emitted from a substance containing air. As thus defined, a clear sound evidently is yielded by percussing any air-containing organ. But custom has restricted the employment of the term clear to denote the peculiar resonance obtained by striking over pulmonary tissue. When, therefore, a clear sound is spoken of, it means a sound having the nature of that of the lungs, or of normal vesicular or pulmonary resonance.

A *tympanitic* sound, on the other hand, is a non-vesicular sound, having the character of that of the intestine. Wherever heard, it indicates the presence of quantities of air in conditions similar to that con-

FIG. 21.

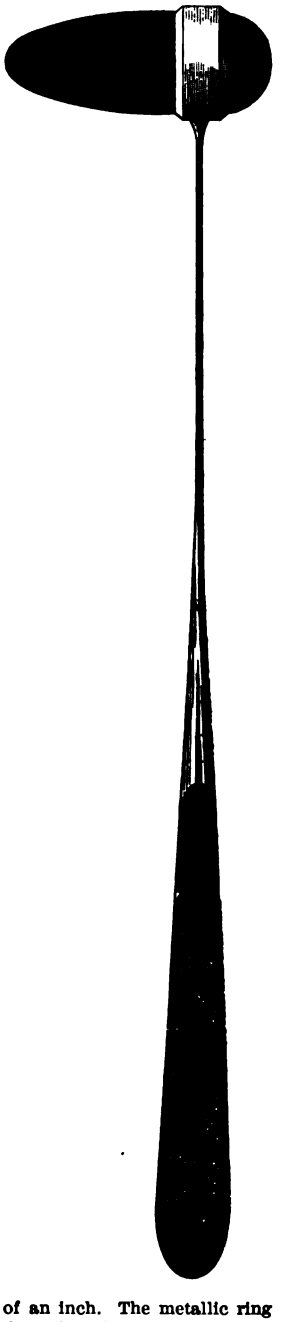


FIG. 21.—A serviceable model of a percussion hammer; not quite natural size. The india-rubber is screwed to the ring, which has a diameter of from five-eighths to three-quarters of an inch. The metallic ring is attached to a steel stem with a very decided spring. The pointed portion of the india-rubber is used to strike within on the plexímeter.

tained in the intestine,—namely, enclosed in walls which are yielding, but neither tense nor very thick. When elicited over the chest, it may be only the transmitted sound of a distended stomach or colon. But generally a tympanitic sound over the seat of the lungs is expressive of emphysema, or of pneumothorax, or sometimes of a cavity, or of œdema of the lungs. Again, as Skoda has taught us, it occurs in moderate pleural effusions above the level of the liquid. The tympanitic sound is distinguished chiefly from the clear sound or pulmonary resonance by its more ringing character and its higher pitch.

If the cavity communicate with a large column of air in the bronchial tube, the note on percussion varies, as pointed out by Wintrich, accordingly as the patient opens or closes his mouth. It is more markedly tympanitic and higher in pitch when the mouth is wide open. Altering the position from a sitting to a horizontal one, when the cavity is partially filled with fluid, Gerhardt has shown changes the tympanitic percussion note, and I have observed it to be markedly altered—indeed, to disappear—on a full held inspiration.[1]

As modifications of the tympanitic sound may be viewed the *amphoric* or *metallic* sound, and the *cracked-pot* or *cracked-metal* sound. The first of these is a concentrated tympanitic sound of raised pitch, and denotes a large cavity with firm, elastic walls. The second is not unfrequently found associated with it. It requires for its development a strong, abrupt blow of the percussing finger while the patient keeps his mouth open. The condition that usually occasions the sound is a cavity communicating with a bronchial tube. It is also met with uncombined with an excavation, as in the bronchitis of children, in pleurisy above the seat of effusion, near a pericardial exudation, in emphysema, and in certain instances of pneumothorax. Indeed, any disorder in which the chest walls remain very yielding, and in which a certain amount of air contained in the lung or pleura is, by sudden percussion, forced into a bronchial tube, will occasion this cracked-metal sound.

In addition to the character of all these sounds, we study their *degree*, or amount of fulness: such changes as are expressed by "more or less," "diminished or increased." Thus, a clear sound may be increased, owing to stronger vibrations and a larger quantity of air, yet not lose its distinctive pulmonary character, as happens often, for instance, when the air-cells are dilated; the sound of the large intestine is fuller, more tympanitic, than that of the small intestine, and so forth.

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[1] Amer. Journ. Med. Sci., July, 1875.

With changes in fulness or volume of sound go hand in hand changes in its *pitch*. Increased volume is linked to lowered pitch, diminished volume to higher pitch; but so is increased tension.

To sum up the chief results of percussion, as above described:

QUALITY, OR CHARACTER OF SOUND.

CLEAR:—Presence of air,—as in the lung-tissue.

DULL:—Solidification or compression.

TYMPANITIC:—Certain amount of air enclosed in a structure or cavity the walls of which are not too tense.

Metallic:—Large hollow space, with firm but elastic walls.

Cracked-metal sound:—Usually a cavity communicating with a bronchus.

DEGREE, OR INTENSITY.

Any of the sounds mentioned may be *diminished* or *increased* in intensity as the conditions which produce them are modified.

PITCH.

Heightened or lowered as amount of air or as tension is altered.

If it be desirable to obtain a more distinct idea of the sound than can be done by the ordinary method of practising percussion, it may be accomplished by resorting to *auscultatory percussion*,—a method that consists in listening, with a stethoscope applied to the parietes, to the sounds elicited by percussion. It is a means of determining with accuracy the boundaries of organs, as of those of the lungs or heart, or of the liver or spleen, and yields particularly good results when carried out with the double stethoscope.

The percussion sound will also be found to vary with the respiratory movement, and useful information may be obtained by the appreciation of the note elicited by percussion while the breath is held after a full inspiration or in a prolonged expiration,—a method of diagnosis which I have introduced under the name of *respiratory percussion*.[1]

As a standard for comparison in disease, the results of respiratory percussion in health must be carefully determined. It will be found that in the normal chest, anteriorly, a full held inspiration increases the resonance, makes the sound fuller, and raises the pitch; but, making allowance for the cardiac region, the resonance below the apices is relatively less increased on the left than on the right side.

[1] Amer. Journ. Med. Sci., July, 1875; see also Friedreich, Deutsches Archiv für klin. Med., Bd. xxvi., confirming these observations.

Posteriorly, we find in the supraspinous fossæ, and on a line towards the spine, that a full inspiration makes the percussion sound fuller and raises the pitch, especially on the right side. In the interscapular and infrascapular regions the tone on gentle percussion is distinctly pulmonary and the pitch moderately high. On the left side an admixture of tympanitic resonance may be detected, particularly in the infrascapular region. The pitch is somewhat lower in the left scapular and infrascapular region than in the right. A full held inspiration elevates the pitch, increases the resonance very much, and makes the difference between the sides less apparent. A held and complete expiration greatly lessens resonance, makes the tone less full, and lowers the pitch on percussion.

Percussion of the Healthy Chest.

The sound elicited by striking a healthy chest differs in accordance with the part percussed. The *anterior* portion renders a clearer sound than the posterior, on account of the slighter thickening of the thoracic walls. But the pulmonary resonance is not, even anteriorly, alike at all parts. The portion of lung above the clavicle yields a sound which becomes somewhat tympanitic as the trachea is approached. Percussion is difficult in this region, as it is almost impossible to apply the finger or pleximeter closely to the surface. Over the clavicle the sound sent forth is clear at the centre of the bone; at its scapular extremity it is duller; towards the sternum it becomes of higher pitch, and mixed with the sound of the bone. In the region bounded above by the clavicle, and below by the upper margin of the fourth rib, the resonance is very marked. In fact, the sound of this region may be taken as a type of the pulmonary sound: it is very clear and distinct, and but little resistance is offered to the percussing finger. Yet a slight disparity generally exists between the two sides. On the right side the sound is somewhat less clear, shorter, and of a higher pitch, than on the left. From the fourth rib downward, on the right side, the resonance of the lung on strong percussion, is found to be slightly deadened; near the sixth rib the perfectly dull sound indicates that the liver has been reached. On the right side, during full inspiration, the liver is pushed downward for the space of an inch or more; and the dull sound on percussion begins, therefore, lower down, and on a line corresponding to the displacement of the organ.

On the left side the heart deadens the sound from the fourth to the sixth rib, and, in a transverse direction, from the sternum to the nipple. This dull sound is lessened in extent during inspiration, and

in cases of emphysema; indeed, under any circumstances in which the lung more completely covers the heart. Lower down, owing to the liver reaching over to the left side, and to the presence of the spleen and a portion of the stomach, the sound rendered on percussion consists of a mixture of the dull sound of the solid viscera and of the clear sound of the lung with the tympanitic sound of the stomach. The latter character of sound predominates when the stomach is empty. Over the upper part of the sternum, to the third rib, the percussion sound is slightly tympanitic; at the lower part, the heart and liver cause this tympanitic or tubular character of sound to give way to a dull sound.

Position exerts some influence on the results of percussion. On exchanging the recumbent for the erect posture, the pitch of the sound on the front of the chest is raised.

At the *posterior* portion of the chest the sound varies materially according to the part percussed. Directly on the scapulæ the sound is duller than between the bones, or than below their inferior angles. Beneath the scapulæ a clear sound is emitted as far as the lower border of the tenth rib; here, on the right side, the dulness of the liver begins. Strong percussion, however, causes the dulness to become manifest higher up. On the left side, below the angle of the scapula, the percussion sound may be tympanitic if the intestine be distended; or it may be rendered slightly dull by the spleen. In and under the axilla the sound is very clear. But on the right side, at the lower border of the sixth rib, dulness becomes perceptible; at a corresponding situation on the left side, the sound is clear or tympanitic from distention of the stomach; and at the ninth or tenth rib, dulness and a sense of resistance to the finger disclose the presence of the spleen.

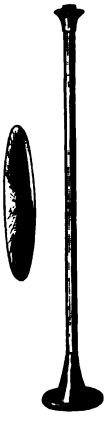
AUSCULTATION.

Auscultation, or listening to sounds, informs us of the play of organs, and furnishes us with the most trustworthy means of studying their action. The method practised by Laennec, the discoverer of auscultation, was the *mediate*, or by the stethoscope. Another method has since his time grown up,—the *immediate*, or the direct application of the ear to the chest. For ordinary purposes, this is the best; but where it is desirable to analyze circumscribed sounds, as in diseases of the heart, the stethoscope is preferable.

Stethoscopes are made of various materials and of different shapes. One of moderate length, with an ear-piece which fits the pavilion of the ear, and with the extremity not too much expanded, is to be preferred. The material is of less importance. I like best those of gun-

metal, introduced by Hawksley. Of late years double stethoscopes have been much employed. The instrument invented by Cammann, of New York, consists of two tubes, the extremities of which are placed into the ears. It has since been modified by making the tubes attached to the ear-pieces of flexible rubber and detachable. But it has also been improved by arranging it to cut off external sounds.[1] A similar kind of stethoscope is the differential stethoscope of Alison, by which each ear receives simultaneously the sound from a different region. It is very little used.

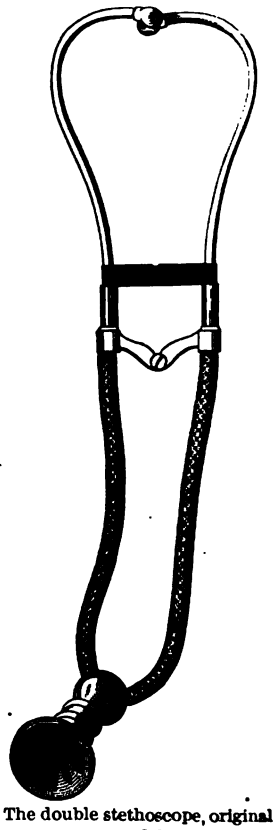
FIG. 22.



Hawksley's stethoscope, with detached ear-piece.

The most recent addition to our means of studying sounds is the *phonendoscope* of Bianchi.[2] It consists of a metallic box about the size of a large watch, with two vibrating plates. Two elastic tubes serve as conductors, and, with a small buttoned rod secured to the lower plate, any point to be specially localized can be examined. Fig. 24 shows the instrument.

FIG. 23.



The double stethoscope, original model.

The phonendoscope is valuable because it is readily applied, and does not produce exaggerated sounds. It is of especial use for the outlining of organs, as a substitute for auscultatory percussion as ordinarily practised. Rubbing the surface with the index-finger over the part to be examined takes the place of percussion with the finger or the hammer. For purposes of comparative auscultation it is also valuable, and several persons can listen at the same time by using different instruments, or by attaching more elastic tubes to one. The phonendoscope is of marked service in studying muscular sounds, and of undoubted value in cardiac diagnosis. While

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[1] Described by Knapp, Medical Record, Nov. 9, 1895.

[2] See Transactions of International Medical Congress at Rome, 1894; Comptes-Rendus de la Société de Biologie, 1896.

all the claims made for it have not been substantiated, I believe it to be a distinct addition to our means of auscultation, and better than the double stethoscope. I have certainly used it to advantage.

In auscultating, the following rules are to be borne in mind:

1. Place yourself and your patient in a position which is the least constraining and permits of the most accurate application of the ear

FIG. 24.

The phonendoscope, natural size; the elastic tubes are, however, much shorter than in the real instrument. The small rod above is screwed on when needed for purposes of minute localization.

or stethoscope to the surface. Above all, avoid stooping, or having the head too low.

2. Let the chest be bare, or, what is better, covered only with a towel or a thin shirt.

3. If a stethoscope be employed, apply it closely to the surface, but abstain from pressing with it. This may be obviated by steadying the instrument, immediately above its expanded extremity, between the thumb and the index-finger.

4. Examine repeatedly the different portions of the chest, and compare them with one another while the patient is breathing quietly. Making him cough or draw a full breath is, at times, of service, especially the former, when he does not know how to breathe.

Sounds of Respiration in Health and in Disease.

The ear applied over the trachea of a healthy person, and subsequently over the lungs, discriminates two dissimilar sounds, which may be severally taken as starting-points.

The first is plainly blowing, both in inspiration and in expiration. It is heard over the larynx and trachea; and in a slightly modified form, as a less intense and hollow sound, at the upper part of the sternum; and sometimes, owing to the closeness of large bronchial tubes to the surface, it is perceived between the scapulæ, on a level with their ridges. It is occasioned by air passing through the tubes, and is known as the tubular or the *bronchial* sound.

The sound over the lung-tissue is different: it is much softer, more gradually formed, of lower pitch, mainly inspiratory, and almost immediately followed by a shorter and far less distinct expiration. This is the *vesicular* murmur,—produced in the finest bronchial tubes and air-cells by their expansion and contraction. The expansion gives rise to the distinct breezy inspiration; the noiseless contraction of the elastic walls of the vesicles and the passage of air back into the smaller bronchial tubes cause the short, indistinct, sometimes almost inaudible expiration. But the vesicular murmur is not exactly alike at different parts of the lungs. It is, as a rule, better marked over the upper lobes than over the lower, and more clearly defined anteriorly than posteriorly. Nor is the sound of the two lungs precisely the same; a disparity may generally be noticed at the apices. Most authors describe the vesicular murmur as more intense on the right side. Investigations instituted to determine this point lead me to agree with Flint that the reverse is the case. More expiration, a higher pitch, therefore more of the bronchial element, is presented by the upper portion of the right lung; but a stronger, more vesicular inspiration belongs to the left lung.

The murmur of the air-cells, then, is the sound which the ear encounters when it is placed over the greater part of the chest. Bronchial respiration is constantly engendered in the tubes of the lung; but, either because it is overpowered by the sounds of the myriads of expanding air-vesicles, or because the pulmonary tissue is a bad conductor for a deep-seated sound, or perhaps because the sound requires consolidated tissue for its perfect production, bronchial breathing is not heard over the chest, except at the very limited space indicated, unless the action of the air-vesicles have been suppressed.

Disease, however, gives rise not only to changes as absolute as suppression of the vesicular murmur and its substitution by a bron-

chial respiration, but also to certain modifications of the murmur, which serve as valuable guides in diagnosis. Thus, the vesicular murmur may be abnormal in its intensity, or in its rhythm, or it may have lost some of the elements of its distinctive character, such as its softness.

Changes in the Vesicular Murmur.—The changes of the murmur which are of importance may be summed up as follows:

Alteration in Intensity	Increased, or puerile breathing; Diminished, or feeble respiration; Absent respiration.
Alteration in Rhythm	Divided and jerking respiration; Alteration of length of expiration relatively to inspiration.
Alteration in Character	Harsh respiration.

Intensity.—An increase of the vesicular murmur is called *supplementary* respiration, or, from its resemblance to the breathing of children, *puerile* respiration. It depends upon an increased action of the air-vesicles; more air, or air with greater force, entering them. The sound is simply a loud, distinctly vesicular respiration; both inspiration and expiration being augmented in duration and loudness, but retaining their relative length.

Peurile breathing is not in itself a sign of any disease. It indicates rather greater activity and energy of the part over which it is heard, which activity makes up for the deficient action of other parts. In this manner effusions compressing one lung, one-sided deposits, or obstruction of the bronchial tubes by secretions, necessitate a supplementary respiration in the healthy portion of the same lung, or in the other.

A diminution of the vesicular murmur, or *feeble* respiration, consists in a lessening of the whole sound without change in its character. But the relation of inspiration to expiration does not remain the same as in health. In the large majority of instances the inspiration suffers most, and the expiration does not diminish in proportion; a circumstance explained by reference to the states which occasion the diminished vesicular murmur. These are varied; but their causes may be reduced to four:

1. Any cause which obstructs the passage of air and prevents it from fully reaching the pulmonary tissue. Foreign bodies lodged in the trachea or bronchi; affections of the larynx; considerable thickening of the mucous membrane of a bronchial tube; its compression, or the accumulation in it of secretions, or its contraction by a spasm,—all diminish the quantity of the air and the force with

which it reaches the vesicles, and hence reduce the strength of the murmur.

2. Deficient respiratory action. This may arise either from general debility; or from impairment of the nervous force, as in paralysis; or from local pain, as in pleurisy or in pleurodynia.

FIG. 25.

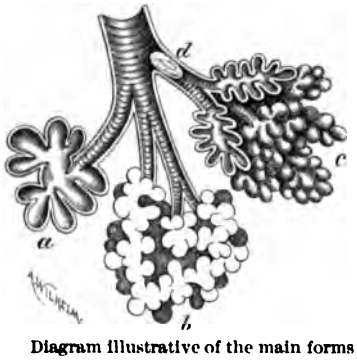

Diagram illustrative of the main forms of feeble respiration: *a*, from distention of the cells in vesicular emphysema; *b*, from deposits in the pulmonary texture; *c*, from a solid body (*d*) lodged in a bronchial tube, which has led to partial, or, in some spots, to complete collapse of the air-vesicles.

3. Causes which interfere mechanically with the free expansion of the air-cells. Pleuritic effusions, by compressing the lung-tissue, will of course diminish the vesicular murmur; so, too, will morbid growths, or malformation of the chest. Comparatively slight deposits in the pulmonary tissue of tubercle or of lymph obliterate some air-cells, and prevent others from unfolding, and, by having impaired their elasticity, diminish their sound. The same loss of elasticity happens in emphysema; the over-distended cells cannot expand much more, they are rigid and more or less fixed; the vesicular murmur is therefore feeble.

4. The respiratory murmur may be imperfectly transmitted to the ear, owing to intervening fluids or solids. To this category belongs the enfeebled murmur so constantly met with in fat persons.

As so many conditions occasion a feeble respiratory murmur, it is only by association with other phenomena that it acquires much importance. Taking the diseases in which the sound is most frequently found, it may be stated that, if a feeble murmur be combined with dulness on percussion, it signifies a tubercular deposit, or a pleuritic effusion: the former, if at the upper, the latter, if at the lower part of the lung. If it be connected with increased clearness on percussion, distention of the air-cells is its cause. A vesicular murmur, feeble throughout both lungs, with the percussion sound unaltered, arises from general debility, or from obstruction of the upper air-passages. Where the feebleness of the murmur is found to change from place to place, it is dependent upon a loose foreign body which is shifting its position in the bronchial tubes. Joined to unwillingness to expand the lung, on account of the pain thereby brought on, feeble respiration denotes pleurodynia or beginning pleurisy.

An absence of the vesicular murmur is produced by the same causes,

carried a step farther, which occasion feeble respiration. Complete obstruction of the tubes by foreign bodies, extensive deposits in the pulmonary tissue, or its compression by large pleuritic effusions, arrest the vesicular murmur. But, practically speaking, there is only one condition in which we are apt to find it entirely wanting, and that is when, associated with flatness on percussion, the presence of a large collection of fluid in the pleura is attested. Extensive deposits in the lung-tissue, tubercular or lymphous, also suppress the sound of the air-cells; but they do not suppress all sound. The noise of the tubes, the bronchial respiration, then takes the place of the vesicular murmur, and denotes the perfect consolidation of the pulmonary tissue.

Rhythm.—The inspiration and the expiration may be altered as regards their rhythm. The inspiration may be broken up into little puffs,—jerking respiration; or both inspiration and expiration may be lengthened or shortened. But neither lengthening nor shortening of the inspiratory murmur has a distinct clinical value; and *jerking inspiration*, met with as it is in spasmodic affections, in hysteria, in pleurodynia, and in tubercular infiltrations, is present under too many different circumstances to have by itself much diagnostic significance. But if limited to the apex, it may serve to excite, or aid in corroborating, a suspicion of tubercular deposit. One modification of the rhythm is, however, of decided importance,—a marked increase in the duration of the expiratory murmur while the patient is breathing quietly.

Prolonged expiration denotes that the air has difficulty in getting out of the lung. It is detained in consequence either of loss of elasticity of the cells, or of an obstruction in the bronchi. The former state may be occasioned by over-distention of the air-vesicles, as in emphysema, or by deposits which impair their contractile power. In the first case, the prolonged expiration is associated with augmented clearness on percussion; in the second, with impaired clearness. Where the prolonged expiration is met with at the apex in connection with dulness it is most often caused by a tubercular deposit.

But a prolonged expiration from tubercular or from any other kind of infiltration is not simply the pure, prolonged expiration of deficient elasticity of the air-cells. It is something more. The solid material conducts a portion of the sound of the bronchial tubes to the ear; and bronchial breathing is nearly always best and earliest perceived in expiration. Thus, a prolonged expiration, when joined to dulness on percussion and to an inspiration still vesicular, is a sound partly vesicular, partly bronchial, and may be interpreted as

consolidation of the lung-tissue; consolidation not sufficient to have obliterated all the air-cells, but sufficient to have obliterated some, and to have impaired the contractile power of others.

The obstacle to the exit of the air may reside wholly in the bronchial tubes. Such is the source of the prolonged expiration when the mucous membrane of the bronchi is swollen. Not only does this condition cause the air to be retained longer in the air-cells, but the resistance to the exit of the column of air brings out more of the bronchial sound. On the whole, then, an accurate study of the expiration is of decided value; and it is of importance to inquire into the expiration separately from the inspiration.

Character.—A distinctive character of the vesicular murmur is its softness. From the moment it loses this, it begins to pass into the bronchial sound. Respiration which is wanting in softness is termed *harsh* respiration, or, to modify slightly a term introduced by Flint, *vesiculo-bronchial*. Harsh breathing is, in truth, a union of the vesicular and bronchial sounds; it is a vesicular sound mixed with some of the qualities of a bronchial sound,—a rough inspiration devoid of all the softness of the normal respiratory murmur, with a prolonged, somewhat blowing expiration. Any affection which, without destroying the murmur of the vesicles, causes the sound in the bronchial tubes to be produced with greater intensity, or to be better transmitted, will occasion harsh breathing. Thus, it exists when the bronchial membrane is swollen, as in bronchitis, and still more frequently in diseases which are attended with compression of the lung-tissue, or with partial condensation, such as some stages of the forms of phthisis or of pneumonia. Being a transition murmur to bronchial, harsh respiration shares the properties of the latter in having its expiration more developed than its inspiration. It is true, the inspiration alone may be harsh, and the expiration not be much changed; but this is uncommon.

Harsh respiration may be confounded with puerile respiration, with sonorous râles, and with bronchial breathing. From the first it varies by its higher pitch, its harshness, its more distinct and blowing expiration; from sonorous râles, with which, however, it often coexists, by the absence of all vibrating or musical character. From bronchial respiration harsh respiration differs merely in degree: it is mixed with more of the vesicular sound, is less blowing in inspiration, and, when produced by condensation, is not associated, owing to the smaller amount of deposit giving rise to it, with so much dulness on percussion.

Bronchial Respiration.—Purely bronchial respiration may ex-

hibit the same modifications as the vesicular murmur in respect to rhythm and intensity. But neither its rhythm nor its intensity is of significance; its character is. To hear well-defined bronchial respiration is, in the majority of cases, to meet with complete consolidation of the pulmonary tissue. It is thus that in extensive infiltrations and in hepatization of the lung we find the bronchial or blowing breathing so marked; particularly so in the latter morbid state, for the most distinctly blowing, or tubular, respiration is heard in pneumonia.

The bronchial breathing encountered in disease resembles more that heard in health over the larynx or trachea than that heard over the larger bronchial tubes. It entirely replaces the vesicular sound, which has for the time being ceased to exist. It differs from the normal vesicular murmur by its higher pitch; by its occurrence equally in inspiration and in expiration; by its blowing character, especially in expiration; and by the pause between inspiration and expiration. Harsh respiration resembles it most; but this, or vesiculo-bronchial respiration, is, as already stated, a transition from vesicular to bronchial breathing.

Whether bronchial respiration be owing, as Laennec taught, to a better transmission of the sound of the tubes through the solid lung; or whether it be produced, as Skoda declared, by consonance, is not of much consequence for diagnosis. The important practical fact connected with this form of respiration is, that it happens when the pulmonary tissue is condensed; this, in the large majority of cases, takes place from exudations or deposits, in a small proportion only, from compression by growths or effusions. At times bronchial respiration is also met with in severe cases of asthma in which the air does not expand the air-vesicles.

A variety of bronchial respiration, at least so far as the quality of the sound determines the point, is that significant sign, *cavernous* respiration. This is essentially a blowing sound; yet it is not always distinct during both inspiration and expiration, being often only perceptible in the one, and mixed in the other with gurgling. It is less diffused, more hollow, and of much lower pitch than ordinary bronchial respiration, and is apt to alternate with gurgling. Hollow spaces of any kind—from abscesses, from bronchial dilatation, from breaking-down cheesy degeneration, from softening tubercle—give rise to it. Its comparatively low pitch may cause it to be confounded with the vesicular murmur. With reference to this it is only necessary to recall that the vesicular murmur is devoid of all blowing quality.

Amphoric respiration is a blowing respiration engendered in a large cavity with firm walls. Its peculiar character is owing to an

echo from the walls of the cavity. It may be humming and of low pitch, or decidedly ringing and metallic. Amphoric or metallic respiration is always indicative of a large cavity; the sound is rarely met with in phthisis; much oftener is it heard over the cavity which is formed between the layers of the pleura, by the entrance of air.

Another variety of breathing connected with a cavity is the so-called *metamorphosing breath sound*, to which Seitz has called attention. It occurs only in inspiration, and consists of a very harsh sound, which lasts for about one-third of the period of inspiration, when it is continued as blowing respiration, attended with metallic echo or ordinary râles. The cause of the phenomenon is the air entering through a narrow opening to reach the cavity. Flint[1] regards this sign as a variety of what he calls *broncho-cavernous* respiration. The sound of expiration in broncho-cavernous breathing is bronchial, high in pitch, and indicates a cavity situated near a portion of consolidated lung. In *vesiculo-cavernous* respiration the cavity is surrounded by comparatively intact pulmonary tissue, and this gives an admixture of vesicular sound.

New or Adventitious Sounds.—These consist of sounds which have no analogue in the healthy state, and which are not, therefore, modifications of the normal respiration. Of this kind are the râles; crackling; the friction sound.

Nearly all *râles*, or rhonchi, are sounds which are generated in the air-tubes by the passage of air through them when contracted or when containing fluid. In the first case are occasioned dry, in the second, moist râles. Râles may occur in inspiration or in expiration, or during both acts. They may obscure or entirely take the place of the natural murmurs. They may have their seat in the upper air-tubes, or in any division of the bronchi. When in the larynx or in the trachea, they are called tracheal râles; of these the death-rattle is an example. When in the bronchial tubes, they are designated bronchial râles; and, as this is their most frequent situation, the term râle means a bronchial râle unless the location be specially indicated.

Dry râles are, for the most part, produced by the vibration of thick fluids which the air cannot break up, and which temporarily narrow the caliber of the tube. When this narrowing exists in the smaller bronchial tube, the sound which results is high-pitched,—*sibilant*; when in the larger, unless the caliber be much altered, it is low-pitched, more musical,—*sonorous*. A similar difference is observed with reference to the moist or bubbling sounds. When the

[1] Lectures on Physical Exploration of the Lungs, 1882.

fluid is thin, whether it be mucus, blood, or serum, and breaks up into large bubbles, large bubbling sounds are occasioned; when it separates into small bubbles, small bubbling sounds are the consequence. The latter, for obvious reasons, generally take place in the smaller tubes.

FIG. 26.

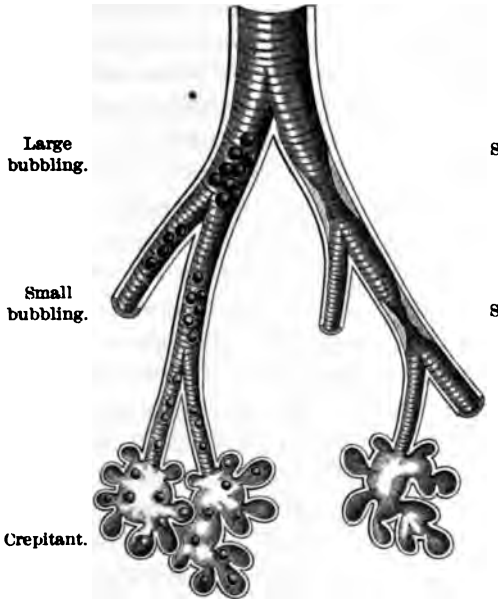

Diagram illustrative of râles. The narrowing in one division of the tube gives rise to dry, the fluid in the other to moist, râles. The râles at the termination of the tube and in the air-vesicles are the crepitant or vesicular râles.

Neither dry nor moist râles are persistent, but vary in intensity, or shift their position, as the air drives the liquid which gives rise to them before it. Dry râles are particularly prone to be dislodged by coughing. When they are uninfluenced by the act of breathing or of coughing, they do not depend upon the presence of secretions, but upon a narrowing of the air-tubes from the pressure of surrounding tumors, or from a fold of thickened mucous membrane, or by a spasm.

It has just been stated that râles are, for the most part, produced in the bronchi by the passage of air through fluids there contained. This is their most frequent seat; but they are not limited to the tubes. Similar conditions may give rise to râles in other places. We find liquids in cavities breaking up into large, sharply defined, bubbling râles, the so-termed cavernous râle,—*gurgling;* or having in cavities of considerable size a ringing *metallic* character; and again, the presence of fluid in the air-cells occasions a minute râle, the *crepitant.*

This vesicular râle, or *crepitation*, is a very fine sound, or rather a series of very fine uniform sounds, occurring in puffs, and limited to inspiration. It resembles the noise occasioned by throwing salt on the fire. Its name indicates its seat. It is caused by the agitation of fluid in the air-cells or in the finest extremities of the bronchial tubes; or, to adopt a view now held by many, by the forcing open during inspiration of the air-cells agglutinated by the exuded lymph. The first stage of acute pneumonia is the state in which this râle is mostly engendered.

The râles, including crackling, may be thus grouped:

- Bronchial Râles.
  - Dry or vibrating sounds.
    - Low-pitched (sonorous).
    - High-pitched (sibilant).
  - Moist or bubbling sounds.
    - Large bubbling (mucous).
    - Small bubbling (subcrepitant).
- Vesicular Râles.
  - Crepitation.
  - Crackling (?).
- Râle of Cavities.
  - Hollow bubbling, or gurgling.
  - Metallic râles.

Crackling is a sign closely connected with râles, and, though its mechanism is undecided, it is regarded as a râle. It consists of a few fine and readily discerned crackling sounds which happen generally in cases of pulmonary tubercle, of which, therefore, they are considered as diagnostic.

The distinction between crackling and the crepitant râle is puzzling. The chief difference is in the number of the sounds. Crackling is a few fine sounds limited to inspiration, and heard commonly at the apex of the lung. Crepitation is a number of fine sounds limited to inspiration, but more diffuse, and heard generally at the base. The sound is similar because the conditions giving rise to it are similar. Both depend upon tenacious fluid or semifluid matter in the ultimate structure of the lung: in the one case it is tubercle or cheesy degeneration, in the other usually the exudation of beginning inflammation. The crackling which indicates softening, as of tubercle,—called by some authors moist crackling, by others clicking,—is a succession of sounds like small moist râles, only less liquid than these, because breaking-up tubercle is not very fluid. When cavities form, and the fluid matter in them is agitated by the ingress and egress of air, the large bubbling, ringing râle of cavities, or gurgling, is occasioned. Dry crackling, moist crackling, and gurgling accord then with the crepitant râle, small bubbling, and large bubbling sounds, and happen in the progressive stages of infiltration and softening of deposits, and generally in those of a tubercular nature.

Pleural friction, or the sound due to the rubbing together of roughened pleural surfaces, consists of a number of abrupt superficial noises heard in inspiration and expiration, rarely in either alone. Its seat is not usually extended, for it is, as a rule, only audible over portions of the lower part of one side of the chest. Sometimes it is so creaking and intense as to be distinctly perceptible to the hand as well as readily recognizable by the ear. But it may be so much like crepitation that even long practice in auscultation will not enable us to determine at once whether the fine sounds we hear are the friction of a roughened pleura, or the vesicular râles of an inflamed lung.

Nor is it, in some cases, less perplexing to discriminate between fine friction sounds and fine moist râles. By the sound alone it is often impossible; concomitant phenomena must be taken into account. A friction sound is mostly confined to a smaller space, and is uninfluenced by cough; while cough changes the position and the distinctness of râles. Yet even this rule is not absolute. A fine friction sound may be temporarily increased during the deep breathing which follows the act of coughing; on the other hand, the influence which cough exerts on the small moist râle is not so great as on the larger bubbling sound. As for the more marked character of moisture which a râle is said to possess, that only aids us in some cases. The features most at variance between the friction sound and crepitant râles are: that the friction phenomena are not strictly limited to inspiration as are the vesicular râles, are not seldom coarser in expiration than in inspiration, are less uniform, and that their seat is more circumscribed. Their production nearer to the ear may assist us, but does not always. The reason why some of the finer friction sounds resemble so closely fine moist râles or crepitation is apparent when we reflect that the irregularities in the pleura may be slight, and be surrounded by fluid which keeps them moistened. Bruen has called attention to the value of making the chest walls immovable.[1] When the chest is fixed, especially at the lower two-thirds, by the hand of an assistant, and the ear or the stethoscope is applied over the doubtful sounds, they will be found to have disappeared if of pleural origin, but to be still discernible if râles.

The creaking or grating varieties of friction are much easier of recognition than the finer forms. Their discrimination from râles is readily affected by noticing the rubbing and harsh character they possess.

[1] Physical Diagnosis.

Auscultation of the Voice.

When the ear is applied to the thorax of a healthy person who is speaking, a confused hum is perceived, most distinct in adults who are possessors of a deep voice, and tremulous in the aged. Now, the normal *vocal resonance*, for by that name the ill-defined vibrations are called, is more marked on the right than on the left side, and corresponds to the vesicular murmur. Over the bronchial tubes a more concentrated sound strikes the ear. This, termed *bronchophony*, accords with bronchial respiration, and, when detected over the lung, denotes, with rare exceptions hereafter to be referred to, the same as bronchial respiration,—increased density of pulmonary tissue caused by pressure or by deposit. Any normal vocal resonance which is augmented passes by degrees into bronchophony, and has a meaning similar to it.

Of the sound known as bronchophony there are several varieties: the *simple bronchophony* just explained,—observed in pneumonia, or in any form of consolidation; the hollow, *cavernous voice*, or pectoriloquy; and the bleating variety, or *ægophony*. The latter, indicative of a thin layer of fluid between compressed lung and the ear, is a sign generally too transitory to be of much diagnostic value; and pectoriloquy, if by this be understood what Laennec meant,—complete transmission of articulated words,—is of no special significance, as it may be met with where no cavity exists. But if the term be applied to a well-defined chest-voice, of hollow character, and heard as such over a comparatively limited space, pectoriloquy is a distinct physical sign, and really deserves the name of cavernous voice. This is particularly true of *whispering* pectoriloquy. Over large cavities the voice is peculiarly ringing and *metallic*. The conditions which produce amphoric or metallic voice are the same as those which occasion any of the amphoric or metallic phenomena. Be the respiration metallic, be the voice metallic, be the rales metallic, they are all caused by a cavity large enough and with walls firm enough to reflect, to echo the sound.

Bronchophony and amphoric voice are instances of increase and change of character of the normal vocal resonance. A *diminished vocal resonance* occurs when the lung is compressed by air or fluid, as in pleuritic effusions, or in pneumothorax; or when it is greatly distended with air, as in extreme cases of emphysema. Clinically speaking, the sign is most often encountered in pleuritic effusions.

The vibrations of the voice may be *felt* as well as heard. The vibration detected by placing the hand over the thorax when the patient speaks, the *vocal fremitus*, is, like the voice, increased by all consolidation of pulmonary tissue, and diminished by fluid or air in the pleura. Its relations to the voice are, however, not uniform; [a]

sometimes with increased density of the lung-tissue there is no increased fremitus, although there is increased chest-voice. In women the sign is valueless; indeed, its main importance is derived from *its absence* in cases of pleuritic effusions. Like the chest-voice, it is most marked on the right side.

Râles, when extensive, sometimes cause a vibration to be transmitted to the chest walls, as do the fluids in cavities. The former phenomenon is called the *bronchial fremitus*, the latter the *cavernous fremitus*. A friction sound that may be felt is designated as the *pleural fremitus*.

The Combination of the Physical Signs, and the Examination of Patients affected with Disease of the Lungs.

In the preceding pages isolated physical signs have been discussed. But if in the investigation of disease we were to trust solely to isolated signs, our conclusions would be incomplete and unsatisfactory. All the methods of physical exploration must be employed, the results obtained compared with one another, and the attending symptoms carefully inquired into and brought into connection with the physical signs, before a diagnosis is made. The manner of investigating by these methods has been detailed; it need not here be repeated. But what may be repeated is, that there are two lungs; that it is incumbent always to explore both, and, as we proceed, to compare the action of one with that of the other.

As many of the signs elicited by the various methods of physical diagnosis depend on the same physical conditions, they may be studied in groups. The following will be usually found to be associated:

Association of Physical Signs.

Percussion.	Auscultation of Respiration.	Auscultation of Voice.	Vocal Fremitus.	Physical Condition.
Clear	Vesicular murmur or its modification.	Normal vocal resonance.	Unimpaired.	Lung-tissue healthy or nearly so; at any rate, no increased density of lung-tissue from deposit or from pressure.
Dull	Bronchial, or harsh respiration.	Bronchophony.	Increased.	Solidification of pulmonary structure.
	Absent respiration.	Absent voice.	Diminished or absent.	Effusion into pleural sac.
Tympanitic	Cavernous or feeble according to cause.	Uncertain; cavernous or diminished.	Uncertain; mostly diminished.	Increased quantity of air within the chest, or air confined in particular points; states commonly due to a cavity or to over-distention of the air-cells.
Amphoric or metallic	Amphoric or metallic.	Amphoric or metallic	Mostly diminished.	Large cavity with elastic walls.
Cracked-metal sound	Cave-		-in.	Generally a cavity communicating with a bronchial tube.

In adults these phenomena are commonly combined. In children, however, their connection is not so constant nor so apparent. Owing to the extreme elasticity of the thoracic walls and the naturally clearer sound of the lungs, the relations of percussion to auscultation are not the same as in the adult. Dulness, even where the condition exists for its production, is rarely as marked; nor is comparison between the two sides of the chest as valuable, since most of the acute pulmonary affections of childhood are more often double than those of adolescence. Auscultation is much more applicable than percussion. The back of the lungs should be invariably examined, and be first listened to. The position, too, in which the child is auscultated should vary with its age. Very young children may be examined either in a lying or sitting posture in the lap of their nurses, or may be held in the arms of an attendant, who is directed to present the different parts of the thorax successively to the ear of the physician. From the cry, when studied with the ear applied to the thoracic walls, we obtain the same indications as from the vocal resonance.

Infants between two months and two years breathe irregularly, and about thirty-five times in a minute. Between the ages of two and six years the average number of respirations in the same space of time is twenty-three. The breathing is also of a different type from that of the adult; it is abdominal, and can be more readily counted by noting the rising and shrinking of the abdomen than by watching the slight movements of the chest.

Before proceeding to the discussion of the symptoms of pulmonary diseases and of the diseases themselves, let us group the latter according to their anatomical seat.

DISEASES OF THE LUNGS AND THEIR COVERINGS.

- **BRONCHIAL TUBES**
  - Inflammation, or Bronchitis;
    - Acute
      - Of large-sized tubes.
      - Of capillary tubes.
    - Chronic
      - Ordinary chronic catarrhal form.
      - Putrid bronchitis.
      - Fibrinous bronchitis.
  - Dilatation;
  - Narrowing;
  - Diseases of bronchial glands;
  - Spasm of muscular fibres or asthma.
- **LUNG-TISSUE**
  - Congestion;
  - Hemorrhage;
  - Apoplexy;
  - Œdema;
  - Collapse;
  - Hypertrophy;

DISEASES OF THE LUNGS AND THEIR COVERINGS.—*Continued.*

LUNG-TISSUE	Inflammation, or pneumonia, in varied forms; Induration; Abscess; Cirrhosis; Gangrene; Emphysema; Tuberculosis, chronic and acute; Pneumoconiosis; Cancer; Deposits, such as syphilitic, etc.; Parasites.
PLEURA	Inflammation, or pleurisy; Empyema; Hydrothorax; Hæmothorax; Tuberculosis; Malignant growths.
PLEURA AND LUNG	Pneumothorax; Perforations and fistulous openings.
WALLS OF CHEST	Pleurodynia; Intercostal neuralgia; Abscesses, etc.

The Principal Symptoms of Diseases of the Lungs.

Of the symptoms about to be mentioned, not one belongs exclusively to pulmonary diseases. We have met with some of them in studying laryngeal complaints; we shall meet with them again in examining the affections of the heart. And in investigating them here we shall not view them simply with reference to morbid states of the lungs, but shall indicate their general relations to diseased conditions, even at the risk of discussing what might in part be more appropriately discussed elsewhere.

The symptoms which it is proposed more specially to sift are dyspnœa, cough, and hæmoptysis.

Dyspnœa.—Dyspnœa means difficulty of breathing. It is accompanied mostly by a sense of uneasiness and suffocation, and by increased frequency of the respiratory act. But increased frequency of breathing may exist without difficult breathing. The respiration may be slower than natural, yet laborious.

Dyspnœa depends upon various causes. Feeble persons are sometimes troubled with it after the slightest exertion. It may be temporarily produced by any bodily or mental excitement. It is observed when the play of the diaphragm is interfered with, and the

lung cramped in its expansion. This is its cause in ascites, in abdominal tumors, and in pregnancy. It may occur in perverted innervation, as in hysteria, or in connection with cerebral affections, from want of power in the respiratory muscles, or it may be due to morbid blood conditions, as in anæmia, scurvy, uræmia, and septicæmia. It is, however, most frequently met with as a prominent symptom of the disorders of the larynx and trachea, or of the heart, and in the various diseases of the lung and pleura, whether idiopathic or secondary. Being common to so many morbid states, it is not diagnostic of any.

Dyspnœa is usually aggravated by position. When the patient lies on his back, the respiration becomes more difficult. The form of dyspnœa in which the sufferer is obliged to remain in the erect posture in order to breathe, is termed *orthopnœa*. This is witnessed in extensive pleural effusions, in pneumothorax, in œdema of the lung, and in affections of the mitral or tricuspid valves.

Dyspnœa may come on in paroxysms, and constitute the only, or certainly the principal, symptom. This is the case in asthma.

Asthma.—Asthma consists mainly in a spasmodic narrowing of the bronchial tubes, caused by contraction of their circular muscular fibres. Its chief symptom is distress in breathing, occurring in paroxysms, and attended with wheezing. These spasms may be preceded by a feeling of suffocation, or they may come on suddenly. The patient wakes up out of his sleep, finds himself wheezing and with a fit of the disease fully on him. He continues to respire with great difficulty, sits upright in bed, or walks about the room gasping for breath. His look is anxious, the face pale, and the color of the lips shows that the blood is not properly aërated. In spite of the struggle to get air into the lungs, the chest moves but little, and when the ear is placed on it, no vesicular murmur is heard, simply the same loud wheezing that is perceptible to the by-standers; or bronchial breathing at the upper part of the chest, or sonorous and sibilant râles are detected, due for the most part to the narrowing of the bronchial tubes, and disappearing with the spasm. These dry râles are chiefly expiratory, and the lungs are very full of air, and displace the diaphragm downward by several intercostal spaces. At the end commonly of some hours the fit passes off with copious expectoration, and as suddenly as it came. But it may last for days, ameliorating in the daytime, exacerbating at night, and only ceasing gradually. Where it frequently recurs it gives rise to marked emaciation.

The exciting causes of these bronchial spasms are various. In some persons there is no apparent reason for the attack; in others it is brought on by the inhalation of irritating fumes or of disagreeable

vapors. In some it is preceded by digestive disorder, or by bronchial catarrh; in others, again, an interruption to the free circulation of blood in the lung, or a disturbance in the sexual organs or in the urinary secretions, seems to occasion it. It is not unusual to find, on closely questioning patients, that for some time prior to the asthmatic paroxysm they have passed a scanty, dark-colored urine. During the attacks Leyden found in the sputum peculiar crystals, farther on depicted. Another interesting fact connected with the paroxysm has been pointed out by Curschmann,—the presence on the turgid, swollen mucous membrane of the bronchioles of a characteristic viscid exudation. This generally shows in the sputum in little pellets that have a spiral structure, very easily discerned by the microscope.

Now, whatever be the exciting agent that calls the bronchial spasm into existence, the symptoms of the attack of asthma are the result of the spasm. Yet asthma is not often a pure neurosis. The seizure itself is the expression of perverted nervous action. But there are generally permanent conditions present, such as disease of the brain or medulla, of the heart, of the lungs, of the ovaries, of the kidneys, of the stomach, or of the nose,—as polypi or hypertrophic rhinitis,—which act as constantly predisposing causes to these seizures, and lead to attacks, either by direct irritation of the pneumogastric nerves or through the medium of the reflex system. Emphysema especially is a fruitful source of spasmodic asthma. Asthma has been noticed to replace other neurotic affections, such as epilepsy.[1]

The detection of the causes inducing an asthmatic fit may be difficult; but the diagnosis of the fit itself is not so. No disease of the lungs or bronchial tubes is likely to be mistaken for it, because no disease of either gives rise to the same symptoms. The dyspnœa of pleurisy or bronchitis is not paroxysmal, nor is it attended with wheezing. Some of the affections of the larynx and trachea bear a nearer resemblance; yet they, too, announce themselves by different symptoms. Asthma may be distinguished from *croup* by the entire absence of fever, and by its lacking the peculiar hoarse voice and cough which appertain to the forms of this malady. The age of the patient is also very different: asthma is as rare in a child as croup is in an adult. *Œdema and spasm of the glottis* differ from asthma by the much more markedly paroxysmal nature of the difficulty of breathing, by the shorter duration of the seizures, and by the absence of the loud and continued wheezing. The sensations of the sufferer, too, indicate correctly the seat of the obstruction. And so they are apt to

[1] Lancet, June 10, 1893.

do in some of the *paralyses of the vocal apparatus*, where noisy dyspnœa happens, and is aggravated in paroxysms. Further, we are aided here by the aphonia, by the inspiratory character of the stridulous breathing, by the absence of chest râles, and by the obvious lesion seen in the laryngeal mirror. A large *goitre* pressing on the trachea may give rise to dyspnœa and to a noisy sound in breathing; but the cause of both is easily traced to the tumor in the neck.

The most deceptive condition is when the *glands of the neck* enlarge suddenly and press on the trachea. I had, some time since, a young man under my care for acute bronchitis. He was progressing favorably, when one day he presented himself, breathing with great difficulty, and each respiration attended with a noise like the wheeze of asthma. I should have regarded him as having been attacked with asthma had I not, in looking at his neck, detected the group of enlarged glands.

Marked dyspnœa may be occasioned by the pressure of an *aneurismal tumor*, or by an *organic disease of the heart*. But the stridor and the persistent difficulty of respiration in the first, aggravated though it may become in paroxysms, and the constant want of breath in the second, are not likely to be mistaken for the wheezing and the paroxysmal dyspnœa of asthma. True asthmatic seizures may both produce and be produced by a disease of the heart. But what is called "cardiac asthma" is not often a spasm of the bronchial tubes: it is usually only a paroxysmal dyspnœa, or a temporary increase of the dyspnœa, dependent upon a decided obstruction to the circulation in the lungs, and not accompanied by wheezing.

So, too, *renal asthma* is only very rarely a true bronchial asthma, being usually an aggravated form of dyspnœa associated with chronic Bright's disease. So-called *thymic asthma* is a severe dyspnœa accompanying enlargement of the thymus gland, and aggravated in paroxysms. It is especially met with in children.

There is a peculiar form of difficulty of breathing connected with a *loss of power in the diaphragm*. When the disorder is fully developed, even the slightest effort gives rise to a feeling of suffocation and to accelerated respiration. The voice is much enfeebled. But the most significant sign of the paralysis is, that during inspiration the epigastrium and the hypochondria are depressed, while the chest dilates; and the converse takes place during expiration. If there be merely a lessened power of the diaphragm, these phenomena are observed only during forced breathing; a paralysis of one-half of the muscle occasions them on one side alone. Duchenne adds another important diagnostic test of a paralyzed state of the diaphragm,—

namely, that if the phrenic nerve be galvanized, the diaphragm acts again with proper strength, and during inspiration the abdomen rises simultaneously with the thoracic walls. To discriminate the cause of the impaired or lost muscular force,—whether this be due to a lesion of the nervous system, or to inflammation of the muscle or of the adjacent textures, whether produced by rheumatism or by lead poisoning, or originating in progressive muscular atrophy,—we have to rely chiefly upon the history of the case. In *rheumatism of the diaphragm*, an absence of the vesicular murmur over the lower portions of the chest; respiration effected by the upper ribs exclusively; tense, hard abdominal walls; want of power to strain so as to aid the bladder or intestines in expelling their contents, with darting, stabbing pain from the spine to the margin of the ribs on each effort to inspire,—have been particularly noticed.[1] In *fatty degeneration of the diaphragm*, which often coexists with a fatty heart, we find, in its last stage, great distress and difficulty of breathing, and death may rapidly follow the embarrassed respiration.[2]

Another form of dyspnœa is the so-called *Cheyne-Stokes respiration*. It consists in inspirations at first short, then deeper and more and more labored, till the paroxysm is at its height; then becoming shorter, and more and more shallow, until the breathing is suspended. The pause lasts from one-quarter of a minute to a minute, when the respiration begins again in the same manner, first faint, then a little stronger, then still stronger, until it reaches its height, when it again subsides in a descending scale, to end in the same stand-still. This kind of breathing is a very bad sign. It is apt to happen when from some cause the supply of arterial blood is cut off from the respiratory centre in the medulla, or from this and the adjacent vasomotor centre. It is rare in diseases of the lungs, much more common in fatty heart, in disease of the aorta, in tubercular meningitis, in apoplexy and affections compressing the medulla, in uræmia, and in sunstroke. It may be found in cases that recover, and be of long duration.[3]

Cough.—Cough is a sudden and violent expiration, having usually for its object the expulsion of some annoying substance from the air-passages. But it may be purely nervous, and unconnected with the presence of any irritating matter in the respiratory organs. There are several kinds of cough: according to the amount of expectoration,

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[1] Chapman, Boston Medical and Surgical Journal, July, 1864.

[2] Callender, London Lancet, Jan. 1867.

[3] As in the case of granular kidney, reported in the Clinical Society Transactions, vol. xxiii., 1890.

a cough is dry or moist; according to its origin, it is laryngeal, tracheal, bronchial, sympathetic, etc.

A *dry* cough is indicative of irritation. This is often seated in the larynx and the trachea, or in their vicinity, or in the bronchi, or in the lung itself. An elongated uvula, and many of the diseases of the nose or the pharnyx, give rise to a dry cough: it happens, too, in pleurisy and in the early stages of phthisis. In disorders of the larynx and trachea the cough is attended with a peculiar shrill noise, or a hoarse sound. But the irritation may not be situated at all in the respiratory system. Affections of the liver, stomach, intestine, uterus, or brain will occasion an obstinate dry cough. It is also produced by dentition, by diseases of the ear, by the presence of worms in the intestinal canal, by disorders of the heart, and by thoracic aneurism. Again, it may be strictly nervous. The brazen cough of hysteria is dry; indeed, nearly all sympathetic coughs possess a dry character.

A *moist* cough may succeed to a dry cough. The moist cough depends, for the most part, on the presence of fluid in the bronchial tubes or the lung-structure. It attends bronchitis with free secretion, œdema of the lung, the more advanced stages of all the forms of phthisis, and pneumonia, when the exudation is breaking up. It is generally accompanied by a free expectoration, which varies in appearance and amount with the morbid state causing it.

Cough is frequently preceded by a sensation of tickling in the larynx, to which the patient is apt to refer his whole disorder. It is much affected by position. Lying down often increases its intensity. Sometimes a cough occurs in severe paroxysms. In various laryngeal affections, in abscess of the lung, in consumption, and in bronchial phthisis, such fits of coughing are observed. But in no complaint are they so constant as in whooping-cough.

Whooping-Cough.—This is essentially a disease of childhood, and the result of an epidemic influence and of contagion. The peculiar spasmodic cough succeeds to a catarrh of more than a week's duration. During the paroxysms the eyes fill with tears, the child's face is injected and anxious, and its whole appearance shows how it is suffering for want of breath. The air in the lungs is expelled by a series of abrupt spasmodic expirations, when a long-drawn inspiration, attended with a whoop, temporarily puts a stop to what appears to be threatening suffocation. The rest is, however, short. The cough recommences, and is again followed by the loud whooping inspiration. It continues in this manner until, after a copious expectoration of stringy mucus, or after vomiting, the paroxysm ceases, and a more

lengthened calm ensues. These fits of coughing repeat themselves at varied intervals during the twenty-four hours. They are very frequent at night. Yet the child's health remains good, in spite of the violence of the attacks and the length of time they are spread over. The spasmodic cough lasts for weeks; the whoop then ceases, the cough loses its ringing sound, and gradually leaves entirely. It is only in comparatively rare instances that it persists, and is followed by the development of tubercles in the lungs; just as it is only in exceptional instances that bleeding from the nose or lungs, petechiæ on the forehead, or ecchymoses of the conjunctivæ happen during the violent coughing. In about one-half the cases the cough is violent enough to produce ulceration of or around the frænum linguæ, from the force with which the tongue is propelled against the teeth. Frequently the ulcer is covered with a grayish exudation; it is never noticed before the paroxysmal stage is well established. Sugar is at times found in the urine. As an early symptom of whooping-cough, photophobia with dilatation of the pupils has been observed.[1] Convulsions are in very young children not infrequent. Whooping-cough is often associated with measles.

An affection of so long duration, marked by such a peculiar sign as a whoop, is of easy diagnosis. Yet there are certain conditions with which it may be confounded. In its first stage, before the characteristic cough sets in, it may be mistaken for *acute bronchitis*. There is, indeed, at this period, no means of distinguishing between the two disorders, except by taking into account the tendency to choking, to flushing of the face, and to vomiting in whooping-cough; for it is only seldom that the cough possesses from the onset a decided ring. And bronchitis is in fact the most frequent complication, or, to state it more accurately, almost an essential element, of the malady. It is usually present in a mild form at the start; it outlasts the paroxysmal stage. At the height of this, a severe attack of acute bronchitis or of broncho-pneumonia may temporarily mask the special traits of pertussis. Again, occasionally acute bronchitis may exhibit paroxysms of spasmodic cough. But the want of the nervous element in the disease, the absence of the whoop and of the recurring flushing of the face as well as of the vomiting, the dyspnœa between the paroxysms, the decided fever, do not permit us to be long in doubt.

A disease less easy to discriminate from whooping-cough is tuberculization of the bronchial glands, or *bronchial phthisis*. It, too, produces a ringing paroxysmal cough. It, too, occurs in children. There

[1] Huguin, quoted in British Medical Journal, Sept. 26, 1891.

is, however, this difference: the enlarged bronchial glands are apt to press on the surrounding parts. This becomes manifest by the engorgement of the veins of the neck, by the lividity and puffiness of the skin, by the difficulty in breathing and in swallowing. The character of the voice, also, may change; and yet there may be no abnormal physical signs in the chest. But often there is dulness on percussion between the scapulæ, where the swollen bronchial glands lie, and impaired respiration in portions of the lung. The symptoms are those of pulmonary phthisis, with which the disease, indeed, may be associated: there are emaciation, and the same loss of strength, the same sweating at night, the same hectic fever, the same tendency to diarrhœa. At times the affection of the glands induces a caseous pneumonia,—in reality tubercular. Now, when we compare these phenomena with those presented by whooping-cough, we miss the whoop, the vomiting accompanying the fits of coughing, the ulceration or tearing of the frænum of the tongue,—a symptom usual, at least, in decided cases,—the epidemic or contagious origin, and the distinct periods, first of catarrh, then of spasmodic cough, then of gradual decline. We see, on the contrary, an affection of more gradual and uniform progress, which often proves its existence by special signs, among which a venous hum, heard when the stethoscope is placed upon the upper bone of the sternum while the child bends back the head, has been particularly noticed.[1]

When emaciation, hectic fever, and marked cough are met with in the last stage of whooping-cough, it is always highly probable that this has been followed by a tubercular deposit, and finding tubercle bacilli in the sputum confirms this view. It is not likely that such cases will be mistaken for those instances of pulmonary consumption in which violent paroxysms of coughing occur. The age, the origin, the history are different. Equally dissimilar are the history and the symptoms in other spasmodic coughs, such as those of hysteria and of some laryngeal affections.

The Sputum.—The consistency of the expectoration varies very much. When it is viscid and tough, it contains a large amount of mucus or muco-pus, and depends generally upon inflammation or a high degree of irritation of the bronchial membrane or of the lung parenchyma. When it is less tenacious, it has far less mucus, and a preponderance of pus. When fluid and full of air, it floats; when dense and without air, it sinks. Fluid sputum forms a homogeneous mass; dense sputum assumes a round or irregularly round shape.

[1] Eustace Smith, London Lancet, Aug. 1875.

When these purulent masses float in a thinner expectoration, we have the coin-shaped or nummular sputum, so common in instances of pulmonary cavities.

The quantity of the expectoration varies greatly in different diseases of the lungs. In the most acute stages, or in spreading inflammations, it is usually small, and increases as the difficulty lessens. In bronchial dilatation, in pulmonary abscesses, especially when they burst, and in the voiding of a collection of pus in the pleura through the bronchial tubes, the amount discharged is very large, and consists almost entirely of pus.

The color of the sputum depends a great deal on its constituents. When mucous, it is white; when muco-purulent, yellowish or yellowish-green; when purulent, generally greenish or of a yellowish-green. It is also tinged by bile, by pigment, and by blood.

Sputum consists chiefly of water, serum, albumin, mucin, nuclein, and many salts, such as the chlorides of sodium and magnesium, the sulphates of sodium and calcium, and the phosphates and the carbonates of sodium, of magnesium, and of calcium. It has an alkaline reaction. In certain diseases, especially in putrid bronchitis and in gangrene of the lung, it contains ferment that acts like the pancreatic ferment.[1]

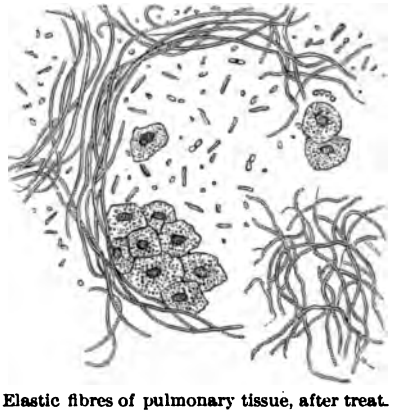
Microscopically examined it exhibits pavement, columnar, and alveolar epithelium, leucocytes, blood-globules, various forms of crystals, such as the slender needles of the fatty acids, fibrinous coagula, bacteria, fungi, and elastic fibres. The alveolar epithelium is mostly of elliptic shape, and often shows little fat drops or pigment particles. The fatty acids and the elastic fibres are encountered in diseases involving disorganization of the lung-tissue. Mould fungi, forms of leptothrix, and sarcinæ have been specially noticed. The latter are smaller than the sarcinæ ventriculi. The fungi are most common in the sputum from cavities, in putrid bronchitis, and in gangrene. The leptothrix masses are readily recognized by their blue stain with a solution of iodine and iodide of potassium.

Elastic fibres in the sputum are very significant; they indicate lung-destruction. They may be found as a bundle of fibres, or in the shape of the alveolar lung structure. In the latter instance they are even a more valuable sign than in the former. Elastic fibres are met with most frequently in tubercular lung-destruction, but they also occur in abscess and in gangrene of the lung, in cavities from bronchitis, and, according to Jaksch, in pneumonia, even where there is no

[1] Stadelmann, Zeitschrift für klinische Medicin, xvi., 1889.

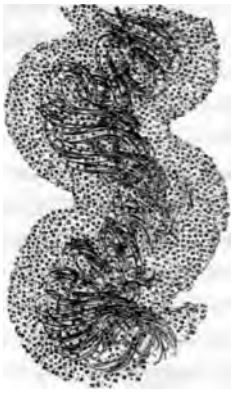
abscess. The most certain way of finding the elastic fibres is to liquefy the sputum by means of caustic soda, or to boil it in a solution of caustic soda or potassa; the particles that fall to the bottom of the vessel can be readily removed and placed under the microscope.

FIG. 27.



Elastic fibres of pulmonary tissue, after treatment with caustic soda.

FIG. 28.



A spiral magnified.

Spirals are structures also possessing considerable significance. They have been studied especially by Curschmann, who traced them to an exudative inflammation of the bronchioles. They are most common in asthma, and bear a close relation to the Charcot-Leyden crystals which are often embedded among the coils. They have also been found in pneumonia. They consist chiefly of a substance allied to mucin, and are large enough to be detected in the sputum with the unassisted eye, though their peculiar structure and the central thread are recognized clearly only with the microscope.

Fibrinous coagula are found in the sputum of pneumonia, of diphtheria that has extended into the lung, but especially in plastic bronchitis, where they furnish a very valuable diagnostic sign. They are moulds of the ramifications of the finest bronchial tubes, whitish in color, of arborescent appearance, and consisting of fibrin. They are small in pneumonia, and do not generally occur in any number; should they be numerous the gravity of the case is greatly increased. They can be seen with the naked eye, or studied with a low magnifying power. In the description of plastic bronchitis a fibrinous mould is depicted.

Different *crystals* can be discriminated only with the microscope. We find cholesterin crystals in the sputum of tuberculosis and in abscess of the lung; the long, thin needles of margaric acid more

especially in pulmonary gangrene and in putrid bronchitis. In the latter disease and in empyema breaking into the lung there have been also noticed by Leyden tyrosin crystals. Crystals of hæmatoidin follow a hemorrhage retained for a time in the bronchial tube. If the blood-crystals be conjoined to cells, they indicate, according to Jaksch, a previous hemorrhage; if any large number of them exist free, they point to a rupture of an abscess from neighboring parts into the lung structure. Uric acid crystals are encountered in the expectoration of gouty patients. The colorless, sharply pointed, octahedral or rhomboidal crystals described by Leyden and Charcot, and named after them, occur in various conditions, as in acute bronchitis; in the blood, intestinal tracts, and bone-marrow of leukæmia; but particularly in asthma. There they seem to have a direct connection with the attack. They are found in the sputum as little, round, yellowish bodies, but require a microscope for satisfactory study. They are soluble in warm water, in ammonia, in acetic acid, and in the mineral acids, and are supposed to be phosphate of ethylenimine.

FIG. 29.

Charcot-Leyden crystals.

Sputum very frequently contains *parasites*. In it may be found the scolices and free hooklets of echinococcus, the actinomycosis fungus, the amœba coli, and others. Sputum full of amœbæ is thin and oily. They generally get into the lung from hepatic abscess following amœbic dysentery. But the parasites of most consequence are the vegetable parasites, especially the bacilli and the cocci. Now, there are many bacilli and cocci that are not linked to any special morbid condition. But these have no particular diagnostic value; the pathogenic organisms are of the greatest importance, and most important is the tubercle bacillus, which is revealed by its significant action towards certain stains, an action that it shares only with the bacillus of leprosy. If the bacillus be exposed to an aniline dye dissolved in an alkali, unlike other pathogenic and non-pathogenic minute organisms, it retains the color on the subsequent addition of decolorizing reagents, such as acids and alcohol. There are many different tests based on this principle. The one of Koch, as modified by Ehrlich, is still, I think, the favorite; though the Ehrlich-Weigert method is also much employed. The Koch-Ehrlich method is as follows: A small drop of sputum is spread very thinly over the surface of a cover-glass, a second cover-glass is laid upon this, and the

two are pressed together and then separated by sliding one over the other. The thin layer on the surface of the cover-glass we select to test is dried by holding it over a gas or an alcohol flame, the side of the specimen being up. The dry sputum is now stained by letting the cover-glass lie for twenty-four hours at ordinary temperature in a saturated solution of aniline oil in water, made by adding the oil drop by drop to distilled water in a test-tube until the mixture becomes turbid, when it is filtered; a few drops of a saturated alcoholic solution of fuchsine, of gentian violet, or of methyl violet, are then added. At the end of this time all the component parts are stained, including the bacilli. The cover-glass is now lifted and immersed for a few seconds in a mixture of one part of nitric acid to three parts of water, until the preparation, previously red, becomes yellowish-green. The preparation is then placed in alcohol of seventy per cent. until no more color is given off; the color disappears, except that of the tubercle bacilli, which are red. We can then counterstain the other parts blue by immersing the cover-glass for a few minutes in a two per cent. watery solution of methyl-blue or of malachite green, unless gentian or methyl-violet has been employed, when Bismarck brown must be used for the background. The cover-glass is then washed in absolute alcohol, dried, and the preparation mounted in oil of cloves or in Canada balsam.

There have been many other processes proposed, among which those of Ziehl-Neelsen and of Gabbett, in which carbolic fuchsine instead of aniline water fuchsine is employed, are much used. The latter is excellent for rapid staining.[1] The cover-slip is kept for from two to five minutes in a cold carbolic fuchsine solution, and then counterstained with a methylene-blue sulphuric acid solution, two grammes of methylene-blue to one hundred grammes of a twenty-five per cent. solution of sulphuric acid. The preparation is then rinsed off in water; the tubercle bacilli show the marked red stain.

As seen when stained, tubercle bacilli are fine rods, absolutely motionless, of the diameter of a human blood-corpuscle, and forming spores of oval outline. Their presence in any number is proof of the existence of tuberculosis; when but few are found it is a question whether they may not have accidentally got into the air-passages.

Another valuable micro-organism in the sputum is the *pneumococcus*, especially the one described by Fraenkel as characteristic of

[1] See, for clear descriptions of the different processes, Schenk's Manual of Bacteriology, translation, London; and Abbott's Bacteriology, 4th edition, Philadelphia, 1899.

pneumonia; it has, however, also been detected in the saliva, in abscesses, in meningitis, and in empyema. Two cocci generally are found together. It is depicted in discussing pneumonia. Let us here examine only the process by which it is best discerned, which, moreover, is a most valuable one in the discrimination of many micro-organisms, the process of Gram.

Gram's decolorizing method makes use of an aqueous solution of iodine and iodide of potassium: one part of iodine, two parts of iodide of potassium, and two hundred and fifty parts of water. The preparation is previously stained in aniline water solution of gentian violet, made in the usual way by shaking up in a test-tube filled with water one to two cubic centimetres of aniline until an emulsion is formed, which is filtered, and to which enough of a concentrated solution of gentian violet has been added to render the liquid of a dark color.

FIG. 30.

Tubercle bacilli.

The iodine solution is then washed out of the tissues; the bacilli or cocci are easily isolated by the stain. The prepared section or cover-glass should be slowly, but completely, warmed in the aniline solution of the gentian violet, either on the water-bath or over the flame, then laid from one to two minutes in the aniline water solution of gentian violet, and subsequently placed in absolute alcohol until the color is discharged. The bacteria show the stain of gentian violet; the tissue may be double-stained red with picrocarmine or other dyes.

This method of Gram is of the greatest value in distinguishing micro-organisms. For instance, it separates the pneumococcus of Friedlaender, which does not stain with it; and the bacilli of cholera, of typhoid fever, and of glanders do not retain the stain.

Hæmoptysis.—Sputa are streaked with blood in bronchitis,

intimately admixed with blood in pneumonia; but it is only when a certain quantity of pure blood is expectorated that the complaint is regarded as hæmoptysis, or hemorrhage from the lungs. Now, a pulmonary hemorrhage may be an idiopathic affection; but it is not often so. It is mostly symptomatic of a grave disease of the lungs or of the heart. It is at times a discharge that takes the place of a suppressed flow of blood from another part of the body, as in vicarious menstruation. Among diseases of the heart, mitral disease is most generally connected with hæmoptysis; among diseases of the lungs, tuberculosis. But it may also occur in gangrene, in bronchial dilatation, in abscess, and in the early stages of pneumonia. We also meet with it in congestion of the larynx, in purpura, in typhoid and typhus fevers, and in arthritic subjects.

When called to a person who has been spitting blood, we have first to solve the question, Where does the blood come from? It may issue from the nose or mouth; from the trachea; from the œsophagus or stomach; it may stream from an aneurism which has burst into the air-passages; or it may be that the lung is bleeding.

When in *epistaxis* the blood, instead of flowing out of the nostrils, flows backward, it is coughed up. But on the patient inclining forward, it will issue from the nose. The color of the blood is not florid; and it can be seen trickling down the pharynx. Inspection is of equal service when the blood comes from any part of the *oral* cavity; especially if it proceed from the gums. Their swollen state, their spongy appearance, and the readiness with which they bleed when pressed, point out at once the source of the hemorrhage.

Loss of blood from the *larynx* and the *trachea*, or from the *œsophagus*, is exceedingly rare; and when it does occur, it is dependent upon some local lesion, such as an ulcer, or the presence of some foreign substance that has been swallowed. By attention to the history, then, we can recognize the cause and the seat of the hemorrhage. The blood itself furnishes no certain mark of distinction. Occasionally the hemorrhage takes place into the interior of the larynx, and only a very small quantity of blood is expectorated. Cases of hemorrhagic laryngitis are usually connected with catarrhal inflammation of the windpipe, with or without ulceration; they are accompanied by severe dyspnœa, and with the laryngeal mirror the blood can be seen trickling down the windpipe.

When blood is vomited from the *stomach*, it is preceded by a feeling of weight and uneasiness in the epigastric region, and sometimes by decided nausea. The ejected matter consists of a dark grumous blood, thus altered by the gastric juice, and is often mixed with

broken-down food. Its dark color is invariable, except where an artery has been laid bare by an ulcer, in which case a sudden discharge of florid blood takes place. There is not commonly more than one act of vomiting; the blood which remains in the stomach passes into the intestines, and goes off with the stools. Hæmatemesis is attended with tenderness at the epigastrium. It is usually symptomatic of an organic affection of the stomach, liver, intestine, or spleen; it may, however, depend upon the swallowing of irritating poisons; or happen in fevers or in scurvy, or as a substitute for suppressed discharges.

The blood which gushes out of the mouth when an *aneurism* opens into the air-passages is red and arterial. It spurts out in jets, and the patient rarely long survives the hemorrhage. Should this not prove quickly fatal, we are seldom at a loss to determine the cause of the bleeding; for we find the physical signs of the aneurismal tumor.

But when the blood comes from the *lungs*, it is of a character, and is connected with symptoms, totally different from any of those just mentioned. The bleeding is preceded by a sense of weight and of uneasiness in the chest. The patient perceives a saltish taste in the mouth and a tickling sensation in the larynx, when suddenly the mouth fills with blood, or, after a slight cough, he expectorates a quantity of light-red and frothy blood. His anxiety becomes great; the skin is covered with a cold sweat; the pulse is quick and bounds under the finger. He spits up more blood, and this continues to come up at varying intervals and in changing quantities all day, or for several days, or even for a much longer period. It is at first pure blood, or mixed with the sputum; is red and not coagulated, and frothy, except when the hemorrhage is very profuse. But after one or two bleedings, the matter which is coughed up contains dark clots, being the blood which has been retained somewhere in the air-passages since the previous attack. The blood is never, at the onset of the hemorrhage, dark and grumous; yet in rare cases it has more of a venous than of an arterial hue. The amount which is brought up at one bleeding ranges from one to two drachms to as many pints; but the quantity that comes out of the mouth is by no means an index of the quantity extravasated. The blood may be effused into the pulmonary structure, and but little be expelled.

After the description above given, it is unnecessary to point out the marks of discrimination between blood ejected from the lungs and blood from other parts. The symptoms are different; the blood itself is different. And listening to the chest detects bubbling sounds in the air-tubes; still, to find these is not requisite for the diagnosis

of pulmonary hemorrhage, and indeed, while the bleeding is going on, the patient's welfare forbids an extended thoracic examination. The bleeding is mostly owing to an affection of the heart or the lungs, and is exceedingly prone to be repeated.

Yet the lungs may bleed frequently without there being an organic lesion within the chest to account for the hemorrhage. I had, some years ago, a patient under my care who had been spitting blood daily for five years. Although enfeebled by the loss of blood, his general health remained good. His lungs and heart appeared to be sound. Another patient had pulmonary hemorrhages at varying intervals for eighteen months. He finally died of exhaustion; but he never presented any physical signs of thoracic disease. In another case that I watched for years, the repeated hemorrhages were found, at the autopsy, to be unconnected with disease of the lungs.

In these instances the hemorrhages recurred often. But we meet with robust persons in whom the loss of blood follows active exercise or exertion, and is not apt to be protracted or to be repeated. In such cases, of which I have seen a number in soldiers sent to hospitals after the fatigue of a long march or the excitement of a battle, simple congestion of the lungs is probably the cause of the disorder.

Except under the circumstances mentioned, hæmoptysis is a grave symptom. It is not dangerous as regards its immediate termination, but dangerous because it is, usually, the indication of a serious malady. Few die as the direct consequence of the hemorrhage, but many die of the disorder of which the hemorrhage is the consequence.

Diseases in which Clearness on Percussion is met with and constitutes a Valuable Sign.

Some of these ailments are acute, others chronic; and nearly all have as their prominent symptom a cough, and are affections, or follow affections, of the bronchial tubes.

Acute Bronchitis.—This is an acute catarrhal inflammation of the bronchial mucous membrane, which occurs idiopathically, or happens as a secondary complaint in the course of fevers, of rheumatism, and of cardiac disorders.

Bronchitis varies considerably according to the tubes involved. The symptoms of acute bronchitis of the *large and middle-sized tubes* are, a sensation of tickling in the throat, soreness or pain behind the sternum and along the lower ribs, a slight oppression in breathing, and a paroxysmal cough. Let us add to these pain in the limbs, coryza, and a fever of moderate intensity, rarely reaching 103° F., and we have the main phenomena met with during the onset and

at the height of an attack of ordinary acute bronchitis. The fits of coughing in the earlier stages are followed by a clear, frothy expectoration, which, as the cough becomes looser and less fatiguing, changes from an almost transparent fluid to a yellowish or greenish sputum. This may be uniform or streaked with blood; it may be small in amount, or in considerable quantity; and it consists chiefly of pus cells and of large, round, alveolar cells with some blood-corpuscles. The fever soon leaves; but long after it has ceased, the patient still has a cough and expectoration, both of which only gradually disappear.

The physical signs may be inferred from the lesions. As there is no condensation of pulmonary tissue, there is no dulness on percussion, the thickening and injection of the bronchial mucous membrane not being sufficient to modify materially the normal resonance. But these conditions must alter the respiratory murmur. They bring out more of the bronchial element of sound, hence more expiration with the coarser inspiration.—in other words, a harsh respiration; or the swelling obstructs the entrance of air into the air-vesicles, and enfeebles the vesicular murmur. Again, new sounds, the râles, are produced; first dry, then moist. This succession of the râles is, however, not absolute, and depends, to a great degree, on the density of the fluid in the bronchial tubes. Dry râles, mixed with moist, may be perceived even in the later stages of acute bronchitis, and long after the febrile signs have ceased. In fact, the tenacity alone of the exudation determines the nature of the râles, and even somewhat their exact character; for every dry râle is not precisely like every other dry râle, nor every moist râle equally moist. With reference to size, the sonorous râles and the large bubbling sounds prevail when the disorder attacks the larger tubes. Sometimes, when the bronchial inflammation is severe and extensive, we find a sound which seems to be neither a dry nor a bubbling râle, but rather a compound of both, —a dry sound, yet not continuous, giving the idea of being caused by the breaking up of fluid. Or, there may be a mixture of the sounds of respiration with the râles, occasioning a peculiar kind of breathing, one in which we can recognize neither a distinctly vesicular nor a distinctly bronchial element, nor a well-defined râle. All these states are dependent upon the amount, and, above all, upon the condition, of the exudation in the bronchial tubes. But they indicate nothing beyond the fact that there is an exudation present which is very large in quantity and tenacious in character. When the sounds are of the indeterminate nature just alluded to, the vibrations produced in the tubes are apt to be transmitted to the parietes of the chest, occasioning with each respiration a marked fremitus.

The diagnosis, then, of acute bronchitis is determined by the cough, the fever, the expectoration, and the signs of clearness on percussion, diffused râles, or harsh respiration. From all those diseases of the lung which result in the consolidation of the pulmonary tissue, such as *pneumonia* and *tuberculosis*, we distinguish bronchitis by the absence of dulness on percussion. Some cases of *acute tuberculosis* on account of the sudden invasion of the malady and the general diffusion of the physical signs, are liable to be mistaken for acute bronchitis; but the different progress of the disorder usually clears up all doubt. Error in diagnosis is more likely to arise from the habit, when the signs of bronchitis have been made out, of not looking farther; forgetting that it is far from always idiopathic, and particularly its frequent association with the eruptive fevers, such as measles and smallpox, with typhoid fever, with influenza, or its occurrence in rheumatism and in malaria.

Capillary Bronchitis.—This is a disease of the aged and of young children. It begins with an acute inflammation of the larger bronchi; or the disorder may from the onset affect the smaller tubes. In either case, signs of obstructed circulation soon manifest themselves; there is lividity of the lips and cheeks, with hurried breathing, a rapid pulse, an anxious countenance, great restlessness, moderate fever temperature, and a cough, followed by viscid expectoration. As the malady advances, the color of the skin and the mucous membranes shows more and more the want of properly aërated blood; the sputa cease with the failing strength; and in old persons delirium and coma, in young children convulsions, mark the closing struggle.

The physical signs are those of ordinary bronchitis, but modified by the seat of the malady. High-pitched whistling sounds, accompanied or superseded by very fine moist râles, denote the smaller size of the tubes involved. The resonance on percussion is clear, or very slightly different from that of health. When materially duller, it indicates that the pulmonary tissue itself shares in the inflammation, or that it has been exhausted of its air and has collapsed.

The parts of the lung which the physical signs prove to bear the brunt of the disease are the lower lobes. In the upper there may be large râles and some fine ones; but it is low down and at the posterior portion of the chest that the fine sounds are most abundant. Yet when the inflammation is extensive, and the accumulation of secretions and morbid products great, quantities of small râles are heard at every part of the chest.

Like the more usual kind of acute bronchial inflammation, capillary bronchitis is liable to be mistaken for *acute lobar pneumonia* and

for *phthisis*. And in the majority of cases the same rules serve for its discrimination; the absence of percussion dulness and the diffusion of the morbid sounds are here again of the utmost value. The rapidity of the attack and the signs of suffocation might mislead into the supposition of the existence of œdema of the glottis, of laryngitis, or of croup; errors in diagnosis which the detection of fine chest râles will prevent.

Capillary bronchitis which really merits the name is a very rare disease, though I believe it to exist. What is called capillary bronchitis is for the most part *catarrhal pneumonia* or *broncho-pneumonia* one of the most common, as it is one of the most fatal, of the diseases of childhood. Like capillary bronchitis, the disease affects both lungs. It is commonly observed in connection with measles, whooping-cough, influenza, or diphtheria; it is especially seen in children previously in impaired health, or scrofulous, or rhachitic. It is apt to be attended by cerebral symptoms,—indeed, it may set in with these,—by rapid breathing and paroxysms of dyspnœa, and by high and irregular fever, ranging between 102° and 105°. As the inflammation is limited to the lobules, it yields but imperfect signs of consolidation. The bronchial breathing is rarely very marked; crepitant râle is not usually perceived, or can scarcely be distinguished from the small bubbling sounds of fine bronchitis; and, from the usual association with inflammation of the fine bronchial tubes, it is in individual cases often extremely difficult to say whether portions of the lung-tissue are consolidated. Theoretically, broncho-pneumonia may be distinguished from capillary bronchitis by the dulness on percussion; practically, this aids but little. Dulness on percussion is in children difficult to elicit; and, again, a dulness may be temporarily produced in capillary bronchitis by collapse of the pulmonary tissue.

Broncho-pneumonia may or may not be preceded by bronchitis of the fine tubes. We may suspect that the inflammation has affected the lobules, if the breathing be very rapid, the fever severe, and the temperature, which is rarely above 102° in the preceding bronchitis of the finer tubes, rise suddenly by several degrees; if the cough lessen as the pneumonia develops; if laryngeal symptoms arise; and if, in addition to râles, not very diffused, spots of dulness, which do not change their seat, and do not disappear under respiratory percussion, be discerned, and plastic pleurisy appear as a complication. On the other hand, when there are early and marked signs of deficient aëration of blood; when the child seems to suffocate from want of power to expectorate; when a multitude of fine dry and moist sounds are heard at every part of the chest, and little or no corresponding

impairment of resonance on percussion is detected,—we know that the capillary bronchi are extensively filled with pus and morbid secretions, and that true suffocative catarrh is threatening life. Capillary bronchitis is a rapid disease; catarrhal pneumonia runs a much slower course, lasting perhaps weeks, and showing a temperature record that is marked by great alternations between morning and evening.

Chronic Bronchitis.—The symptoms and signs of chronic bronchitis are not very different from those of the ordinary form of acute bronchitis. The duration of the complaint and the absence of fever, except during marked subacute or acute exacerbations, are the chief distinguishing elements. Yet the cough, although on the whole chronic, is far from being constant. It may disappear almost altogether, and then reappear with more than its previous severity; and this state of things may go on for years, undue exposure and change of season aggravating the disorder.

The sputa vary, even more than in acute bronchitis, in tenacity and quantity. There may be merely a small quantity of yellowish matter expectorated in the morning, or an almost continued flow from the bronchial tubes,—*bronchorrhœa*. The physical signs differ accordingly. A harsh or feeble respiration, and few or many, either dry or moist, râles, are present, in conformity with the state of the bronchial mucous membrane and of the secretions. The sound on percussion is clear, and this, with the diffusion of the signs discerned on auscultation, is of great importance. Excessive secretions somewhat impair the pulmonary resonance, but only temporarily; for with the shifting secretions shifts the very slight dulness.

One of the most important points in the diagnosis of chronic bronchitis is to attend to the manner in which it arises. It may follow a seizure of acute bronchitis, or be the result of recurring attacks of subacute character; it may appear as a primary affection, or it may follow the exanthemata; or, again, it may complicate some previously existing disorder, as Bright's disease, rheumatism, lithæmia, gout, psoriasis, or eczema, and be directly traceable to the constitutional taints of these maladies; and its symptoms will vary and be influenced by those of the general malady to which it is subordinate.

In the ordinary idiopathic malady the general health, as a rule, suffers but little. In some instances, however, emaciation takes place, and the disease simulates phthisis. This is particularly the case in the bronchial affections among knife-grinders and coal-miners, also in those of granite-masons, of sandpaper-makers, of flax-dressers, and of potters. The resemblance becomes still greater when superadded

bronchial dilatation and fibroid induration of the lung produce physical signs like those of pulmonary consumption.

A chronic *catarrhal inflammation of the mucous membrane of the nose* may be mistaken for chronic bronchitis, with which, indeed, it may coexist. But when occurring uncombined, there are no râles in the chest or altered breathing-sounds indicative of disorder there, though there may be a cough, from the throat being also affected. The secretion, too, from the nose is very copious and of muco-purulent character, the upper part of the nose looks somewhat flattened, and the sense of smell is impaired,—not one of which signs is met with in chronic bronchitis. A minute inspection of the nasal membrane or a rhinoscopic examination is of most value.

It seems almost unnecessary to speak of the differential diagnosis between chronic bronchitis and *rose cold* and *hay asthma*. The coexistence of marked signs of irritation of the eyes, the nose, and the throat; the appearance of the distressing affections at a particular period of the year; the fixed time in which they run their course; their occurrence in those of neurotic constitution and having an irritable nasal mucous membrane; the almost instant relief on leaving the regions where the attack has been brought on and on reaching favorable localities; the depression of the nervous system; and, on the other hand, the less decided signs of bronchial affection,—clearly distinguish the maladies.

We meet occasionally with a form of bronchitis in which the expectorated matter is solid. This *plastic bronchitis* presents all the usual signs and symptoms of bronchial inflammation. It may be chronic or it may be acute. It is most frequently chronic, with occasional acute or subacute exacerbation. The disease extends in this way over weeks, months, or even years, and is apt to end in complete recovery. But in its acute form it is a complaint of great danger and accompanied by much dyspnœa, and has led to death by suffocation.[1] Males, as we find by looking at the cases which Peacock[2] has collected, are more often attacked than females. The same carefully collated observations show that the disorder affects more commonly the upper than the lower part of the lungs. As regards the physical signs, Fuller,[3] who has met with a number of well-marked examples of the complaint, states that there is weakness or entire absence of breathing over the affected portions of the lungs, and that, from at-

[1] Andral, Clinique Médicale.

[2] Transactions of the Pathological Society of London, vol. v.

[3] Diseases of the Chest.

tending collapse, complete and rapidly developed dulness on percussion may ensue. But the only absolutely diagnostic phenomenon is the peculiar membranous material expectorated. In form this may be either in thin shreds, or moulded into an accurate cast of a bronchial tube and its ramifications. The expectoration of the firm bodies is sometimes attended with copious hæmoptysis. The casts consist of layers of fibrin in which leucocytes and alveolar epithelium are embedded. Leyden's crystals and Curschmann's spirals may be found. The disease is most apt to occur in the spring months.

FIG. 31.

Cast from a case of plastic bronchitis.

The little, round, solid pellets which consumptive patients, or even some persons in good health, cough up from time to time are the result of a plastic bronchitis on a limited scale; but in a certain proportion of chronic cases decided plastic bronchitis and tuberculosis are associated. A kindred disease to plastic bronchitis has been described as "bronchiolitis exudativa." The sputum is grayish and very tenacious, and full of spirals which come from the bronchioles. Gradually increasing dyspnœa and attacks of asthma are prominent symptoms.[1]

Another variety of chronic bronchitis is *putrid bronchitis*. This may happen in connection with bronchial dilatation or with chronic pneumonia, or without these conditions; occasionally it appears after a suppurative pleurisy which has broken into the lung. There is fever with irregular temperature; at times chills occur. The distressing cough is followed by a copious half-liquid sputum, extremely offensive, and containing little yellowish plugs, the so-called Dittrich plugs. The peculiar odor is thought to be due to a micro-organism, especially to a short, slightly curved bacillus described by Lumnitzer.[2] Cases of putrid bronchitis may be mistaken for gangrene of the lung; but the odor is different, and they lack the physical signs of lung-destruction and elastic fibres in the sputum. We must, however, bear in mind that putrid bronchitis may terminate fatally by induced pneumonia or pulmonary gangrene. Sometimes it produces death by metastatic abscess of the brain.

[1] Curschmann, Deutsch. Arch. für klin. Med., Nov. 1882.

[2] Wien. Mediz. Presse, May, 1888.

Emphysema.—A distention of the air-cells is a frequent sequel of chronic bronchitis. It may happen in only one lung; but the air-vesicles of both are usually distended. The effect of this is to obliterate some of the capillaries, and to interfere with the flow of blood through the lungs. From this proceed the feeling of constriction and the dyspnœa, the anxious look, the bluish lip, of emphysematous patients, and the tendency the disease has to produce dilatation or dilated hypertrophy of the right side of the heart.

Emphysema is essentially a chronic malady; but in its course subacute attacks of bronchitis occur which much augment the difficulty of respiration. The embarrassment in breathing is, indeed, the most prominent of the symptoms. It is not so much the difficulty of getting air into the lung, as it is of getting it out, which annoys the patient. He breathes as if he had no object but that of forcing the air out of the pulmonary tissue. And this task is often aggravated by spasmodic narrowing of the bronchial tubes; hence it is very common to meet with the loud wheezing of asthma in those whose air-cells are permanently dilated. In long-standing cases of the disease the patient looks cachectic, is cyanosed, the shoulders are rounded, the chest is barrel-shaped, and dropsy of the feet is noticed. There may be also a chronic cough, which may be dry and occur in paroxysms of marked intensity.

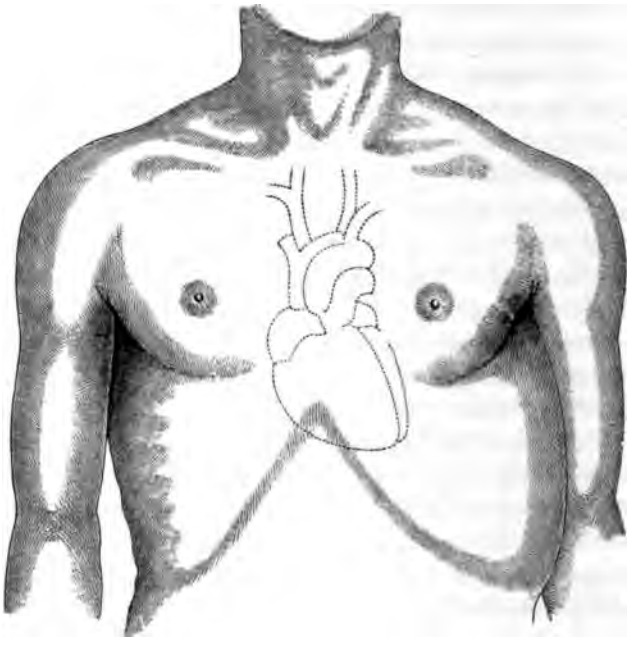
The physical signs of emphysema are easily deducible from the pathological conditions. The distention of the lung-tissue explains the great prominence and fulness of the chest, and the displacement of the liver or heart. The ringing clearness on percussion—at times almost tympanitic in its character—and the increased resistance to the finger have the same cause. Nor is it difficult to understand how the loss of elasticity in the dilated air-cells will give rise to an unchanged note on respiratory percussion, to prolonged expiration, and to a feeble inspiratory murmur. If bronchitis coexist, the signs on auscultation are necessarily somewhat altered. The respiration is harsh, or intermixed with dry and moist râles. The former especially assume great prominence, and are heard as sonorous, or still oftener as sibilant, râles, during the prolonged and labored act of expiration. Occasionally a crackling sound is heard in emphysema.[1] When the emphysema is partial, all these signs are limited; when it is more general, they are diffused.

If the upper lobe of the right lung or the lower lobe of the left,

[1] Gerhardt, Berlin. klin. Wochenschr., March 12, 1888.

which, according to Louis,[1] are the parts most frequently affected, be emphysematous, the visible local bulging might mislead into the idea of the prominence being due to an aneurismal tumor, or to the presence of fluid in the pleural cavity. Any doubt will, however, be dispelled by a careful examination of the chest. The dulness over an *aneurismal tumor*, its pulsation, and its sounds, are different from the

FIG. 32.



Appearance of the chest in a patient suffering from a high degree of emphysema. The heart is displaced. The other physical signs are extreme percussion clearness; a feeble, hardly audible inspiration; a very prolonged expiration.

exaggerated clearness on percussion and the changed respiratory murmur of an emphysematous lung. *Pleuritic effusions* produce a bulging at the lower part of the thorax. But, although there may be a very clear, or rather a tympanitic, sound above the fluid, the absolute dulness over it shows that the prominence of the chest is not caused by distended air-vesicles. When the emphysema is extended and general, displacement of the liver or heart results; and this, taken in connection with the dilatation of the chest and the dyspnœa, may cause the disease to be mistaken for *pneumothorax*. The differences are pointed out in the discussion of this complaint.

[1] Mémoires de la Société Médicale d'Observation, tome i.

We shall only here add that the affection of the heart, the torpid, displaced liver, and the presence of albumin in the urine, in emphysematous patients, may call away attention from the primary pulmonary cause.

An effusion of air may take place into the areolar tissue uniting the lobules. There are no physical signs peculiar to this *interlobular emphysema;* they are exactly the same as those furnished by dilatation of the air-cells, except that a dry friction-sound and a large, dry crackling, both of which occur occasionally in vesicular emphysema, are much more common. Its suddenness and the external emphysema which follows are specially indicative of the disease. The latter is detected under the jaw, or at the base of the neck, and yields a peculiar crepitation. Yet the extravasation of air into the areolar tissue of the neck is not a constant attendant. Besides, the possibility of a crepitating swelling in the neck being due to a rupture of the bronchial tube or of the larynx must be borne in mind.

The rupture of the air-cells which gives rise to interlobular emphysema is brought about by any severe effort, by violent coughing, by laughing, or by the throes of parturition. It has also been known to happen in the course of pneumonia or of pulmonary hemorrhage, and to have caused sudden death. Its most frequent association is with whooping-cough.

A *compensatory emphysema* is met with when distention of the air-cells takes place in the unaffected lung or in an unaffected lobe. It generally occurs at the anterior margins, and is developed by the high tension in the air-vesicles that have to do more duty. It is chiefly found in extensive pleural effusion, in pneumothorax, and sometimes in pneumonia. The physical signs are those of ordinary emphysema.

In all the disorders which have just been treated of, the resonance on percussion has been dwelt upon as a most valuable sign. Before proceeding to consider the diseases in which dulness is encountered, a few words may here find their place on a morbid condition in which clearness rapidly gives way to dulness, and dulness changes quickly back into clearness. As, moreover, the complaint to which I allude—*collapse of the lung*—has a close connection with bronchitis and emphysema, its consideration is at this time fitting.

The chief cause of collapse of the lung, or post-natal atelectasis, is accumulations in the bronchial tubes. No air can enter the air-vesicles; the residual air in them is gradually exhausted, and the disordered portion of lung is reduced to a state as if it had never breathed. But, although in the majority of instances this post-natal

atelectasis is brought about by catarrhal secretions in the bronchial tubes which cannot be expectorated, any want of power to fill the cells of the lung with air may lead to their collapsing. In some of the typhoid forms of acute and chronic diseases, in the pulmonary congestions of the aged and enfeebled, and in those occurring just prior to death, large portions of the lung-tissue may collapse simply from inability to breathe with sufficient force. We also meet with collapse of the lung in whooping-cough, in compression of the lung from pleural effusion, and in rhachitis.

The physical signs of collapse are not satisfactory; the symptoms vary with the conditions producing the disease. Neither voice nor respiration is characteristic. The most usual physical sign is dulness on percussion, with an absence of all respiration, or with a blowing sound, which is faint and not so distinct as in pneumonia. The dulness is not so great, may be changed during respiratory percussion, and in cases dependent upon inspissated mucus may disappear suddenly when the obstructing cause is removed. Yet collapse of the lung is at times a state of long duration. Should a pneumonic process affect the collapsed portion, the dulness is stationary, and we are apt to find the high but variable temperature of broncho-pneumonia. Under ordinary circumstances the temperature is normal or subnormal.

After collapse the breathing becomes very difficult. The patient makes intense efforts at inspiration; owing to the non-expansion of the lung during these efforts, the ribs move inward and recede, instead of moving outward as in ordinary breathing. This sign, the suddenly increased dyspnœa, and the appearance of dulness in special areas, unaccompanied by marked bronchial breathing, are, in a case of bronchitis, the most trustworthy indications that collapse of the lung-tissue has taken place. Yet where the collapsed lobules are small and scattered through the lung, these signs are not at all present, and the diagnosis is uncertain. The dulness is wanting; and the peculiarity in inspiration may not be observed.

When collapse affects a large portion of lung, it much resembles *lobar pneumonia*. The fever, the absence of retraction of the chest wall, the crepitant râles, the tubular breathing, distinguish this, and bronchophony is much more marked. How nearly collapse resembles *broncho-pneumonia* has already been indicated. The diminution in volume of portions of the chest, the shifting character of the physical signs, and the speedy re-entrance of air into parts that had shown signs of condensation, are the most trustworthy points in diagnosis. In *pleural effusions* the distinguishing signs are the flatness on percus-

sion and the absence of breath-sounds, of bronchophony, of fremitus; besides, we do not find the retraction of the chest walls, and the extremely rapid, superficial breathing.

Diseases in which Dulness on Percussion occurs.

The diseases of the lungs in which dulness on percussion is met with are all those in which compression or consolidation of the pulmonary tissue takes place. Especially do we find dulness, and the physical signs which accompany it, in the phthises, in pneumonia, and in pleurisy.

Phthisis.—Phthisis presents itself in a chronic and in an acute form. The chronic variety is by far the most frequent. It is essentially "the consumption," which is such a scourge to the human race. In by far the greatest number of instances this consumption is linked to tubercular disease. And although we can recognize a non-tubercular form, I shall, unless it be otherwise specified, use the term phthisis as meaning tubercular disease.

Beginning usually with a short and insidious cough, with a feeling of lassitude, and a decline in general health; attended at times from the onset with a pain in the affected lung and a somewhat quickened circulation; or giving the first indications of its existence by the occurrence of a hemorrhage,—the disease becomes fully established, with symptoms which hardly need a detailed description. The harassing cough; the disturbed digestion; the steadily augmenting debility; the short breathing; the exhausting night-sweats; the hectic fever; the deceptive blush which this imparts to the cheek; the increased lustre of the eye; the singular hopefulness; the temporary improvements; the relapses; and the greater vividness of the imagination, so strongly contrasting with the waning frame,—are phenomena with which sad experience has made not only every physician, but many a fireside, familiar.

The most constant of all the symptoms are the hemorrhage, the cough, and the emaciation. The *cough* is at first dry, or followed by a frothy expectoration. As the disease advances, the sputa thicken. They become greenish in color, streaked with yellow, and "nummular," consisting of large greenish masses of a rounded form, which do not sink in the cup containing them, but float imperfectly in a thin serum. This expectoration is, however, by no means pathognomonic; it is occasionally encountered in chronic bronchitis. In the last stages of consumption the sputa are often homogeneous, and have a grayish, purulent aspect. Examined microscopically, they show alveolar epithelium, pus-cells, exudation corpuscles, and elastic

tissue, the most distinctive of which is the elastic tissue of the alveolar walls. Yet the only absolute sign in the sputum is the bacillus tuberculosis. Its presence bespeaks tubercular disease, its absence, on several examinations, is a strong argument against the existence of this affection. The numbers found in the sputum bear a direct relation to the extent and gravity of the complaint; in arrested tubercle they become very few or disappear. In lung destruction from syphilis or from chronic pneumonia, in the non-bacillary form of fibroid phthisis, in cavities from bronchial dilatation, in gangrene of the lung, the bacillus is not observed. But failure to find the bacillus is not as valuable and conclusive evidence as finding it; yet a few of the bacilli may be met with in the sputum from accidental lodgement in the air-passages.

In rare instances, the cough remains slight throughout the malady; but generally it is a distressing feature, and is particularly worrying at night. Sometimes its violent paroxysms bring on vomiting. But vomiting and other gastric symptoms occur irrespective of paroxysms of coughing. In truth, anorexia, nausea, and vomiting are often very prominent and early symptoms, and may exist where no obvious lesion of the gastric mucous membrane is found; dilatation of the stomach attending the dyspeptic symptoms is not uncommon. Some shortness of breath is usual; dyspnœa is rare. Early anæmia, with increase of the blood-plates, is another frequent symptom.

Among the less constant symptoms of pulmonary consumption are a troublesome and rebellious diarrhœa connected with catarrhal inflammation, with fistula in ano, or with tuberculosis of the bowel, chronic laryngitis and chronic pharyngitis, hypertrophy of the mammary gland, more common in men than in women, and the red line around the border of the gum. In some persons this gingival line is a mere streak; in others it is more than a line in breadth; in none is it a certain indication. A sign which has a much more definite connection with tubercular disease of the lungs is the appearance of the nails. The end of the finger is somewhat clubbed; the nail is curved, prominent in the centre, depressed at the sides, its surface slightly cracked, its appearance bluish. A similar nail is, however, seen in chronic pleurisy and in diseases of the heart. The laryngeal symptoms are apt to be a very distressing complication, and mostly end, no matter how they begin, in tubercular laryngitis. This, and the laryngoscopic appearance of the ulcers have been described when treating of laryngeal diseases.

Fever is a very constant and significant symptom of pulmonary tuberculosis. Indeed, the temperature may be greatly elevated for

several weeks before we find physical signs indicative of the deposition of tubercle, or of an undoubted increase in the already existing deposition. Furthermore, the rise in the body heat closely corresponds to the activity of the deposition of tubercle. If the temperature be decidedly and permanently elevated throughout the day, there is active deposition. When the temperature is normal, the deposition in the lungs has ceased, and the tubercular process is arrested or retrograding. It may be also normal or even subnormal in very chronic cases.

The morning temperature in tubercular phthisis is often higher than the evening temperature, though we frequently see the reverse. Very generally the maximum temperature is reached in the afternoon; sweats occur in the evening, and there is a drop of two or three degrees towards morning. The temperature chart of the hectic fever may simulate that of a remittent or an intermittent fever, and the frequent occurrence of chills and the sweats make the resemblance still closer. In the last days of the disease the temperature may fall greatly.

The thermometer has been made use of in another manner in the diagnosis of tubercular consumption. Peter[1] calls attention to the advantage of local thermometry. A surface thermometer is applied firmly in front of the chest in the second intercostal space, and if the temperature is higher there than on the other side, or than normal, it is because there are tubercles underneath. In beginning tuberculosis the increased local heat is in proportion to the extent of the lesions. In health the temperature of the chest walls is about 36° C. (96.8° F.); it may rise in tubercle to 37° C., or more, and in consumption with cheesy degeneration still higher, surpassing the general fever heat of the body.

The symptoms which precede a fatal termination are various. Patients may go on failing for years; or an intercurrent attack of acute tuberculosis, of pneumonia, of tubercular meningitis, or of tubercular ulceration of the intestines, may at any time result in death.

The tendency of tubercular matter is to soften and destroy the textures among which it is infiltrated. It may undergo, at any period in its course, a retrogressive development, by shrivelling up, or by passing into a calcareous state. When situated in the lungs, it seeks the apices by preference; it is rarely limited to one lung, although one lung is usually the most diseased, and often at the beginning of the malady is alone affected. Tuberculosis is not merely a local com-

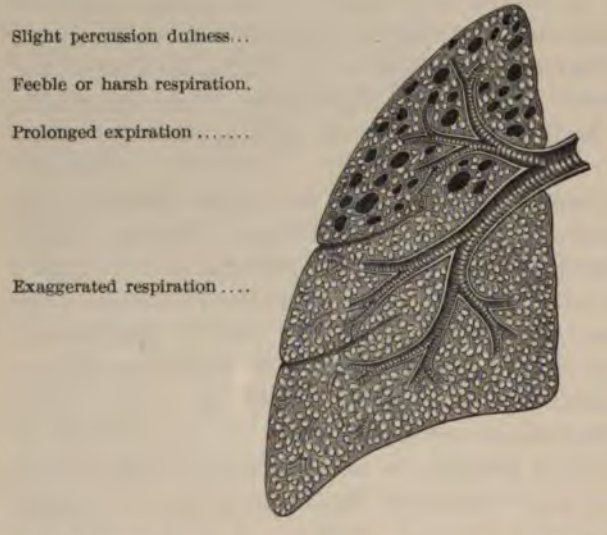
[1] Clinique Médicale, tome ii., 1879.

plaint, but stands in connection with a peculiar, tainted state of the constitution, whether this be produced by infection from the products of the bacilli or not; hence the symptoms of phthisis are not solely the expressions of the condition of the lungs.

In accordance with the laws affecting tubercular depositions, we have three stages of phthisis, which are to be borne in mind when examining the *physical signs:*

1. Incipient stage, or beginning deposition;
2. More complete deposition, occasioning consolidation;
3. Stage of softening and of the formation of cavities.

FIG. 33.



Beginning infiltration; masses of tubercle have accumulated, but the intervening lung-tissue is still healthy.

1. A few scattered tubercles do not change the normal percussion resonance; nor do they appreciably alter the natural breath-sounds. But as soon as the deposit is sufficient to impair the elasticity of the lung-tissue or to increase its density, a relative loss of clearness on percussion on one side, and modifications of the vesicular murmur, such as feeble or jerking inspiration, or a prolonged expiration, may be ascertained. The dulness is readily detected by percussing the patient with his mouth open and during a fixed expiration, or the difference between the two sides becomes very manifest during held inspiration,—in other words, respiratory percussion will aid us. To find the dulness at the upper part of the chest posteriorly, the position

of crossing the arms and clasping the shoulders is very advantageous. In a certain number of cases, with the slight dulness on percussion and the changed breathing is associated a blowing sound in the subclavian or in the pulmonary artery. A murmur is, indeed, at times present in the pulmonary artery long before any other physical indication of tubercle is discernible. All these physical signs may be accompanied by râles of various kinds. What makes them significant is, that they occur at the upper portion of the lung, whether anteriorly or posteriorly. If, therefore, any modification of the vesicular murmur, or any adventitious sound limited to the apex, exist; if there be a slight dulness on percussion above or under the clavicle, or in the supraspinous fossa; if this coincide with flattening of the anterior surface of the chest, especially on one side, with defective expansion of the thorax and shortness of breath, with a cough, and falling off in general health,—the diagnosis of beginning tubercular disease is almost positive. But these signs possess now less value to us than formerly, for the detection of bacilli would be of greater import than any or all of them.

2. As the infiltration advances, the signs become more decidedly those of consolidation. Greater dulness on percussion at the upper portion of one or of both lungs, scarcely influenced by respiratory percussion; more resistance to the percussing finger; stronger vocal resonance; a sinking in of the side most affected, and often soreness to the touch over the diseased part; a very harsh murmur; or, when the infiltration surrounds the bronchial tubes, a distinct blowing respiration,—are all present in varying degree, and all denote consolidation. And chronic consolidation at the apex has, in the large majority of instances, but one interpretation,—phthisis. In the second stage, as well as in the first, we often meet with superadded signs of bronchitis which occasionally mask the respiratory sounds, with friction-sounds from local pleurisies, or with fine crackling. We may also encounter a whiffing murmur, the so-called *cardio-pulmonary* murmur produced by the beat of the heart against the pulmonary texture, and especially heard in inspiration.

3. The diseased organ now passes into a state of softening, or rather some portions of the lung begin to soften, while others remain indurated, and in yet others fresh infiltration takes place. Moist crackling or persistent moist râles indicate that softening has begun. The broken-down material may be expectorated, and the malady for a time be stayed; but such is not often the case. The area of the softened mass widens; cavities form; and in addition to the moist râles, to the physical phenomena of the second stage, and to the in-

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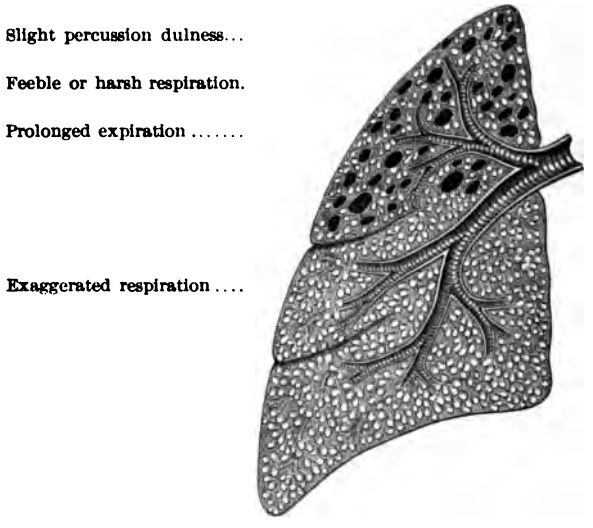

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creasing debility, night-sweats, and hectic, the signs indicative of a cavity are noticed. Prominent among them are the cavernous voice, especially in whispering, and the hollow breathing. But the hollow, cavernous respiration may be caught only in expiration, or it may be temporarily superseded by very large bubbling sounds,—gurgling.

FIG. 34.

Cavities of various sizes.

Again, over small or over deep-seated cavities none of these sounds may be perceived; and, in truth, even when they exist, their limitation to a particular locality is an element in the diagnosis of a cavity almost as important as their presence.

The results of percussion over an excavation are not always the same. They depend much on the thickness and the state of the walls of the cavity. If dense, percussion yields a dull sound; if thin, a tympanitic, or its varieties, a cracked-pot or a metallic sound. If only a certain amount of indurated tissue intervene between the cavity and the surface of the chest, a singular sound, a mixture of dull and tympanitic, is produced. If healthy lung-tissue form the walls of the excavation, the sound is clear, or nearly so. Moreover, in all cases the pitch and, to some extent, the character of the sound are changed by percussing over the cavity while the mouth is kept open. When it is shut, the sound elicited is of lower pitch. On inspiratory percussion, the previously tympanitic or mixed sound becomes dull. Another sign by which we may judge of the existence

PLATE II

it be on the left side, and if alterations of the vesicular murmur correspond to it. When the dulness is not discernible, we have to depend on the history of the case, especially as to family, likelihood of exposure to infection, the occurrence of blood-spitting, the limitation of the physical signs to the apex, the persistency of the cough, and the falling off in general health, out of proportion to the local lesions.

Where the deposition is at all extensive, an erroneous diagnosis of bronchitis is with ordinary care impossible, unless, as is always highly improbable, phthisis should be complicated with *emphysema*, or the tubercles be quiescent, and so diffused as not to impair the resonance on percussion. Under the latter circumstances especially, the occasional tympanitic character of the sound over the seat of the tubercular deposition is liable to be misconstrued into increased clearness on percussion, and into a disproval of the existence of phthisis. When tubercle and emphysema coexist, the percussion note may really be pulmonary and like that of healthy lung; the respiratory sound becomes much feebler; generally, too, the dyspnœa is increased; the temperature is higher than in pure emphysema. But the most certain sign would be the tubercle bacilli in the sputum.

A difficult diagnosis may be at times the distinction between chronic bronchitis and the *phthisis of old people*. This, indeed, often happens in a latent form, and is very slow in its development; the temperature may be normal or subnormal. Besides the microscopic examination of the sputum, auscultation alone is of much value, since the chest remains resonant on percussion, owing to the dwindling of the muscles of the thorax, the ossification of the ribs, and the rarefaction of the lungs.

In the stage in which the signs of consolidation become well defined, phthisis may be mistaken for any of those conditions that occasion the physical signs indicative of greater density of the lung-tissue, and that are accompanied by cough and by loss of flesh. Such are particularly pneumonic consolidation, pleuritic effusion, and cancerous deposits.

Chronic Pneumonic Consolidation.—Chronic pneumonic consolidation, or chronic pneumonia, gives rise to many manifestations which simulate consumption. These are cough, emaciation, and the local signs of chronic condensation,—increased voice and fremitus, sinking in of the chest wall, feeble inspiration and prolonged expiration, or a fully developed bronchial respiration. But in pneumonic consolidation the history usually points to an antecedent acute affection; the health is not so much impaired; there has been no hemorrhage,

although the sputa at times may have been streaked with blood; and the dulness on percussion and the other physical signs of consolidation are, for the most part, perceived over the lower lobe of one lung. In many of these cases interstitial fibroid changes ultimately take place in the lung, and we thus have a chronic interstitial pneumonia, which allies the cases closely to fibroid phthisis. Yet it is clinically convenient to keep them apart, as the consolidation may slowly disappear, and the retraction of the chest and other features of fibroid phthisis as ordinarily seen are not present.

This position of the physical signs is of great importance. Yet there are two sources of fallacy which may arise. On the one hand, tubercles may, by way of exception, be seated in the lower lobe; on the other, chronic pneumonic induration may affect the apex. When an infiltration of tubercle takes place in the lower lobe, its distinction from chronic pneumonic consolidation is very difficult. Our surest guides are attention to the pathological law which teaches that consumption is not met with in an advanced state in one lung alone, and the examination of the sputum for bacilli. Not finding these, and the absence of serious general symptoms, will determine the real nature of the case when an inflammation of the upper lobe has resulted in its persistent induration. I adduce a few instances, by way of illustration:

A gentleman was under my care for years, in whom, after pulmonary inflammation, signs of condensation remained in the upper part of the right lung. He did not suffer at all, except from attacks of acute bronchitis, to which he was very liable. During these he lost flesh; but when they passed off he rapidly regained it. He had a chronic cough, but it was very slight. After the lapse of a number of years I lost sight of him.

In another case, with a similar history, I found dulness on percussion, prolonged expiration, and a friction-sound limited to the apex of the right lung. There had been a continuous cough, but very little constitutional disturbance, and no hemorrhage. The abnormal signs lasted for a year, and then almost disappeared under a succession of blisters, and the cough ceased. In yet another patient, a man seventy-five years of age, the dulness at the right apex had for years remained stationary.

In all instances of doubt between chronic induration and tubercular disease, important information is drawn from watching whether the physical signs undergo changes in the hitherto healthy portions of the lung. To the presence or absence of the bacillus tuberculosis in the sputum the greatest weight must be attached.

A great and complicating difficulty in the differential diagnosis grows out of the circumstance that tubercular disease may be developed in a lung in a state of chronic induration. We find persons in good health seized with inflammation of the lung, which is followed by persistent consolidation, and in the course of time by tubercular phthisis. Indeed, many of the reported cases of tubercle affecting primarily the lower lobe of the lung are, in reality, cases of tuberculosis following chronic pneumonic consolidation. The history is usually as follows. A person in all respects healthy is attacked with an acute pulmonary affection. He recovers from it, but with a trifling cough, with a persistent dulness on percussion, and with feeble respiration, heard over a portion of one of his lungs. He continues ailing, though not positively ill, when, without any apparent cause, after a time varying from a few months to years, his cough increases, the expectoration augments greatly and becomes decidedly purulent, the temperature rises, and he emaciates rapidly. Hemorrhage may or may not happen; profuse night-sweats occur; and the physical signs, which have been stationary for a long time, now begin to change. The dulness extends; and, instead of the enfeebled respiration, a harsher, blowing respiration is perceived over the affected part, and moist crackling and the signs of a cavity follow. If doubt still exist as to the nature of the malady, the advance of the disease will clear it up. True to the laws of tubercle, a deposit takes place in the lung previously sound, and not at the lower portion, but at its apex.

At all stages a minute examination of the sputum will tell us when the bacillar infection takes place. It is supposed by many that the tubercle bacilli have existed in the lung prior to the inflammatory disease, or may, indeed, have caused it. But this is not often borne out by the clinical history. It is more likely that the bacilli have lodged in the damaged organ. Cases of the kind with the cheesy changes in the lung and the disintegrating products of the inflammation form the variety of phthisis that was not long since asserted to be a special disease, *pneumonic phthisis*, but which we no longer doubt to be only a clinically somewhat different variety of tubercular affection.

These remarks apply almost equally whether the original seizure was a croupous pneumonia or a catarrhal pneumonia. Yet there are some special points which the chronic consolidation attending a *chronic catarrhal pneumonia* exhibits. In the first place, the history of a preceding acute catarrhal attack is clear, or there have been a series of attacks, after one of which the lung was left solid, and since which the patient has been prone to take cold, and is easily put out of

breath. Now, he may come under our observation in the midst of one of these broncho-pneumonic seizures, and we may watch him for months with the signs of consolidation over a portion of one lung, whether at base or apex, or with affected points, often symmetrical, in both; further, there are night-sweats, fever with decided evening exacerbation, diarrhœa. Gradually these urgent symptoms yield; he gets about, but a spot or spots of consolidation in one or both lungs do not go away for a long time; or the chronic catarrhal pneumonia may remain as such, or pass into pneumonic phthisis, which really means tubercle. When this happens, great variation between morning and evening temperature, simulating a malarial fever, increasing cough and dyspnœa, marked sweats, decided emaciation, announce the event: while the physical signs show extending dulness, crackling and fine moist râles, over the affected spots or in parts not previously diseased, and ultimately cavities. At all stages repeated examinations of the sputum for tubercle bacilli are of decisive value.

Chronic Pleurisy.—A persistent cough attended with emaciation and with dulness on percussion is common to chronic pleurisy and to phthisis, and is a cause of many errors. But the seat of the dulness at the lower part of the thorax; its much more absolute character; the almost entire cessation of all breath-sound; the diminished or absent vibration of the chest walls when the patient speaks; the dilatation of the affected side,—are in striking contrast with signs most manifest at the apex, with the distinctly prolonged expiration, with the râles and the evidences of beginning softening. Nor are the symptoms of a pleuritic effusion as grave as those produced by phthisis. Even where the fluid filling the chest is pus, we do not find hectic fever so intense, emaciation so great, or night-sweats so constant and exhausting; and the patient coughs less, and never spits up blood. In those cases of chronic pleurisy in which the side, instead of being dilated, is retracted, the diagnosis is more difficult. Attention to the seat of dulness being at the lower part of the chest, to the diminished respiration, voice, and fremitus, and to the shrinking affecting only one side of the thorax, will, however, serve as the foundation for a correct conclusion.

Tubercle may complicate pleuritic effusions. We suspect this by the occurrence of hemorrhage, and by the marked emaciation and hectic. We can only be sure of it by finding signs of deposit on the non-affected side, and by tubercle bacilli in the sputum. Tubercular pleurisy may be a one-sided as well as a primary disease. It is not always accompanied by effusion. There may be only great and irregular thickening of the pleural membrane attended with variable

fever, with coarse friction, with much pain, and with or without bacilli in the scanty expectoration. Chronic *double* pleurisy is very apt to be associated with a tubercular affection of the lungs, but it may be rheumatic, or may occur without obvious cause.

Pulmonary Cancer.—Cancer of the lung shares with tubercle the cough, night-sweats, hemorrhage, gradual wasting, as well as the signs of pulmonary consolidation. But cancerous formations are usually limited to one lung. Only one side of the chest is flattened or distended. Over the cancerous lung the percussion dulness is great. There is either loud, blowing respiration, or, if the mass have compressed or obliterated a bronchus, enfeebled or absent breathing and absent tactile fremitus. We find no râles; but all the signs of consolidation are more perfect than in tubercle. Owing to a cancerous deposit in the mediastinum, the dulness at times extends beyond the median line. Paroxysmal dyspnœa, enlargement of the clavicular lymph-glands, and prominence of the large veins on the chest and arms are common. Fever is generally absent.

Cancer in the lung may soften; yet the signs of softening are rarely as manifest as they are in tubercle. The sputa are purulent, or like currant-jelly or prune-juice, and show no characteristic bacilli. Further, a cancerous tint of the skin may be present; and cancerous tumors in other parts of the body become absolute evidence in favor of a deposit in the lung being cancerous, since, with very rare exceptions, cancer and tubercle do not coexist. The character of the pain must be also taken into account. In tubercle, it is transitory and shifting; in cancer, it is much more constant, and much more severe.

Syphilitic Disease of the Lungs.—Syphilis may occasion a specific form of bronchitis, preceding the syphilitic eruption; or produce gummata, which may soften and be eliminated, and which form in the lungs towards their periphery and base; or give rise to chronic interstitial pneumonia of the base. When syphilis manifests itself in the pulmonary structures, it produces most of the phenomena of phthisis. The chief differences are, that the nodules affect generally only one lung, most frequently the right, and principally the base or the lower part of the upper lobe; that they remain circumscribed, not spreading to the surrounding textures; and that they occasion, as a rule, neither hæmoptysis, nor fever, nor night-sweats, nor decided emaciation, nor marked cough or râles, but dyspnœa out of proportion to the local disease. The most common physical signs are dulness on percussion, deficient fremitus, altered vesicular breath-sounds, and obvious sinking in of the supra- and infraclavicular regions; in

some instances signs of destruction of the lung are found. Still, the syphilitic affection can be distinguished with certainty only by the history of the case, by the thickening of the periosteum of the head of one or both clavicles, and the perichondrium of one or more of the upper cartilages, with frequently a tumefaction of the soft parts between them and the skin, and by substernal tenderness. In all cases we must be careful that the thickening at the upper part of the chest walls and the altered resonance thus occasioned be not looked upon as signs of a tubercular consolidation; and as regards the tenderness, pain on pressure is met with at the lower part of the sternum in a large number of phthisical cases.

Syphilis of the lung may also be associated with syphilitic lesions in other organs, especially in the larynx, and we may find considerable cough, with emaciation, diarrhœa, and albuminuria. But even then there are no night-sweats and fever attending the emaciation, the great debility, and the marked dyspnœa. The diagnosis of syphilis has been made by microscopical examination of the sputum, finding nucleated granular cells, shrivelled nuclei, spindle-cells, and remnants of a finely striated stroma.[1] To the absence of tubercle bacilli in doubtful cases great weight must be attached. Fibrous pleurisy and pleuritic effusions are comparatively frequent; even small cavities occur in the lung.[2] In rare instances syphilis of the lung runs an acute course, simulating acute pneumonic phthisis.

The preceding diseases are most likely to be confounded with the stages of consumption prior to softening and the formation of cavities. Next let us review those affections which, like phthisis, occasion the signs of excavation, and which, therefore, may be mistaken for its third stage: they are, chiefly, bronchial dilatation, abscess, and gangrene of the lung.

Bronchial Dilatation.—A dilatation of the bronchial tubes takes place in two forms: either the tubes are uniformly dilated and like the fingers of a glove, or else they form cavities by undergoing a saccular enlargement. The former variety furnishes the symptoms and physical signs of a case of chronic bronchitis attended with copious expectoration. The percussion clearness may be slightly lessened, owing to the condensation of the surrounding pulmonary tissue; the respiration may be more strictly bronchial; but otherwise both symptoms and signs are those of chronic bronchial inflammation. In the

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[1] Sokolowsky, Deutsche Medicinische Wochenschrift, Sept. 12, 1883; Cube, also Guntz, quoted in Schmidt's Jahrbuch, No. 6, 1882.

[2] Satterthwaite, Boston Medical and Surgical Journal, June, 1891.

globular form of dilatation we meet with all the sounds of tubercular excavations: the hollow, blowing respiration; the hollow, well-transmitted voice; gurgling; even metallic tinkling. In the acute cases, Wilson Fox[1] has observed the metallic quality of the râles to be very distinctive. Yet all these phenomena are in strange contrast with the almost unimpaired health, and with the non-occurrence of hemorrhage, of night-sweats, and of emaciation. Still hemorrhage does happen in a certain proportion of the cases. Pain, Lebert has shown, is among the early manifestations of the disease. The temperature is normal, except during acute or subacute attacks of bronchial inflammation.

Thus, when we find the signs of a cavity, and when the general symptoms do not indicate profound constitutional disturbance, we may suspect a bronchial dilatation. This suspicion becomes a certainty, if the cavity be at the middle or the lower portion of the lung, if the resonance on percussion be but little impaired, and if the slight dulness is not increased by inspiratory percussion, and, for the most part, follows, and does not precede, the auscultatory signs of a cavity. We find further evidence in the stationary character of the physical signs: for months they do not change. They are often associated with unilateral interstitial pneumonia or pleurisy, and with retraction of the chest. The expectoration of bronchial dilatation is more abundant than that of consumption, is apt to be purulent, acid, of oily appearance, and in chronic cases fetid, suggesting, indeed, at times, the existence of gangrene. It does not contain tubercle bacilli, and shows elastic fibres only if there be ulceration. As regards the cough of dilated bronchi, it is persistent, and only at times relieved by expectoration, which varies in copiousness according to the size of the sac, and chiefly occurs after a spell of coughing in the morning.

Skoda[2] describes, as a peculiar physical sign present in sacculated bronchial dilatation, a large and coarse crackling, called by him the large bubbling, dry crepitant râle. In a case which came under my observation, the diagnosis was made by this auscultatory sign. The patient, a boy aged twelve years, had swallowed a bone, which lodged in a bronchial tube and gave rise to bronchitis and bronchial widening. He died subsequently of acute meningitis, and the bone was found firmly embedded on one side of the globularly dilated bronchial tube.

[1] Treatise on Diseases of the Lungs and Pleura, London, 1891.

[2] Percussion and Auscultation.

Bronchial dilatation is observed, as in the instance just mentioned, after impacted foreign bodies. It is also met with after whooping-cough, after long-standing chronic bronchitis in which the bronchus has been weakened by inflammatory changes, and in connection with cirrhosis of the lung. But there are many cases to which Granger Stewart[1] has particularly called attention that are due to atrophy of the bronchial wall, and that probably result from a constitutional defect. In these primary cases the disease comes on insidiously.

Pulmonary Abscesses.—Abscesses of the lung may form in the course of acute pneumonia, but are not then likely to be mistaken for chronic phthisis. Different is it with abscesses which are developed three or four months after an attack of pneumonia, and where the lung-texture has remained partially consolidated. I have seen not a few examples of chronic induration of the lung terminating in this way. A man who was shot through the lung was seized, soon after the injury, with inflammation of that organ. Percussion dulness and blowing respiration continued at the lower part of the left lung. One day, after exertion, he suddenly expectorated a considerable amount of pus. The signs of a cavity were detected at once; but they subsequently disappeared, and perfect recovery took place. In another case of pneumonia, the disease in like manner lapsed into a chronic state. Five months after the acute attack, the evidences of an excavation became manifest at the edge of the right scapula, and existed there for two months; then, so far as physical signs could prove, the cavity closed. Instead of the hollow, blowing respiration and gurgling, only a somewhat roughened vesicular murmur was perceived.

Such is, however, not always the termination. The abscess may grow larger and larger, until the entire lung is destroyed; amphoric percussion note, amphoric respiration, amphoric voice, and, at times, metallic râles, being the physical signs observed.

Lung abscesses differ from *bronchial dilatation* in not being permanent and fixed. They have this in common with *tubercular excavations*,—they change. They increase like these; but, further, they do what tubercular cavities do not, they decrease. Their physical signs are in every respect like those of all cavities, and vary with the size of the excavation. Sometimes metallic respiration and voice may be heard over it; or perforation of the pleura produces the signs of pneumothorax with effusion. In fortunate instances the pus is expectorated, or the abscess opens externally, and a cure is thus established. But very large abscesses are apt to wear out the patient.

[1] Twentieth Century Practice, vol. vi.

Hectic fever and occasional hemorrhage attend them; yet neither is so constant a symptom as it is in consumption. The sputa are usually copious, purulent, full of elastic tissue, and fetid, differing in this respect from the expectoration of phthisis, which is only temporarily fetid, if the secretions decompose in the cavities. Again, abscess of the lung may be distinguished from tubercular disease by being ordinarily situated at the base of the organ; by its following pneumonic consolidation, although there are exceptions to this rule, chiefly in septic conditions; by the occurrence of copious expectoration being often sudden; but especially by its limitation to one lung. The other lung remains healthy. It may enlarge, and its murmur be more distinct; but the sounds denote its texture to be normal.

Abscess of the lung is not infrequent in suppurative diseases of the nose, or larynx, or œsophagus. It is still more common from embolic infection. The small amount of constitutional disturbance which pulmonary abscesses sometimes entail is remarkable, and the physical signs of a large cavity are in strange contrast with the regular pulse, the almost undisturbed breathing, the slight cough, and the healthy complexion.

What has been called "dissecting pneumonia," a suppurative inflammation starting mostly in the peribronchial tissues, dissecting the lobules, and subsequently destroying the parenchyma, leaving nothing but the bronchial ramifications and vessels, has symptoms that are in the main those of abscess, of which, indeed, it forms a variety. The absence of fetid breath and of fetid sputum distinguishes it from gangrene.[1]

Pulmonary Gangrene.—This disease also yields the signs of an excavation. It occurs either as diffused or as circumscribed gangrene, after pneumonia, especially aspiration pneumonia, or typhoid fever, after wounds of the lung, from blows on the chest, from poisoned blood, diabetes, pressure of an aneurism, or from emboli in the pulmonary tissue. The symptoms are: great prostration, dyspnœa, a very pale face, a quick pulse, hemorrhage, emaciation, and a cough, followed by profuse purulent sputa of a greenish or brown color. But nearly all these symptoms happen also in phthisis. What is characteristic of gangrene is the extreme fetor of the expectoration and of the breath. The sickening odor is not perceived during each act of breathing, but mainly after coughing, and, as it were, in jets. It is the symptom by which, especially if taken in connection with the

[1] See an elaborate paper by Hutinel and Proust, Arch. Gén. de Méd., Nov. 1882.

signs of breaking up of the pulmonary tissue and the sputum, gangrene is with certainty recognized. The cavity is found in only one lung, and generally at its lower part. This is of aid in discriminating between phthisis and gangrene; but it does not distinguish between a gangrenous excavation and a simple abscess of the lung. The only positive proof of gangrene of the lung is, as just stated, that the signs of breaking down of the pulmonary tissue are accompanied by a disgusting and more or less persistent fetor of the expectoration and of the breath; sometimes a sickening, faintly sweetish smell, sometimes fecal, oftener that of putrescence. I say persistent, because local gangrene, on a small scale, occurring around tubercular cavities or in bronchitis, may give rise to temporary extreme fetor of the breath. But it is only temporary, and therefore not liable to lead to fallacious inferences. The expectoration may be fetid in cases of bronchial dilatation or of abscess of the lung, but is never brownish, as is not uncommon in gangrene; and neither it nor the breath has the peculiar gangrenous odor. In rare instances pleurisy with fetid effusion may occasion a fecal smell of the expectoration and breath, which is gradually lost.[1] The fetid sputum of fetid bronchitis is not associated with any signs of breaking down of the lung.

Yet in considering the diagnosis regarding *bronchial dilatation* we must not overlook the fact that, as Dittrich and Traube[2] have shown, this bears a marked relation to gangrene. Decomposition takes place in the secretions retained in the bronchial dilatation, and ulceration of the coats may ensue, leading to a gangrenous process in the surrounding tissue. Now, as just mentioned, the sputum even in bronchial dilatation may become fetid. As, moreover, it, like gangrenous sputum, may present a dirty greenish-yellow color, and separate on standing into three distinct strata, of which the uppermost is frothy though dense, the second serous, and the third dense, containing pure pus and detritus; as, further, we meet in both affections with little solid masses of particularly offensive odor full of fat and fine needle-shaped crystals of margaric acid,—we may have to depend, for a differential diagnosis, on finding with the microscope pigment grains and masses of elastic tissue.

Pulmonary Actinomycosis.—This rare disease resembles tubercular disease of the lung in presenting cough, fever, wasting, and a mucopurulent expectoration. The attending fever is of irregular type, sometimes like that of typhoid fever, more generally like hectic fever.

[1] As in the case reported by William Moore (Dubl. Quart. Journ., May, 1865).

[2] Gesammelte Abhandlungen.

The physical signs are mostly those of tubercular deposit. The absolutely distinctive feature is finding the ray fungus in the sputum. Besides the lungs, other parts of the body may be involved, such as the jaw, the alimentary canal, and the subcutaneous tissues.

With reference to other affections which are sometimes mistaken for pulmonary tuberculosis, owing to emaciation and an attending cough, such as intermittent fever, anæmia, dyspepsia, chronic diarrhœa, chronic laryngitis, and chronic pharyngitis, the physical signs are different, and an examination for tubercle bacilli is conclusive.

In the remarks on the diagnosis of pulmonary consumption, the complaint has been assumed to be progressive; in rare instances it *retrogrades*. The signs by which such retrogression can be discovered are not very fixed. In those cases in which many tubercles undergo a cretaceous transformation, calcareous particles are coughed up; the signs of softening cease; fibroid changes take place in the affected lung; the apex flattens; and a feeble murmur with prolonged expiration, or a harsh respiration with slight dulness on percussion, is all that remains to indicate that tubercular disease has existed. The cough stops, and flesh and strength return.

We meet occasionally with instances in which the physical signs of an infiltration into the lung-tissue depart with tolerable rapidity. They occur in those who have a decidedly scrofulous aspect, enlargement of the glands of the neck, or a scrofulous inflammation of the eyes. In accordance with the acknowledged identity of scrofula and tubercle, we are forced to admit that the disease in the lungs is tubercular. Yet the connection with the enlarged lymphatics; the circumstance that the diminution in size of the glands is often followed by increased pulmonary deposits; that these depositions are very beneficially influenced by treatment; that they disappear sometimes altogether, or only reappear months afterwards; that hemorrhage is not among the symptoms,—all make it a question whether there be not a *scrofulous disease of the lung* independent of a tubercular, one pursuing more the course of an external scrofulous disease, one, moreover, which presents a much more favorable prognosis than ordinary consumption. Among scrofulous children cases like these mentioned are not uncommon. The disorder certainly differs from the ordinary forms of pulmonary tuberculosis, and it is not bronchial phthisis. It does not present the paroxysmal cough, the signs of pressure on the trachea or the large bronchi, and the dull sound on percussion between the scapulæ, which are the common accompaniments of enlarged and tuberculous bronchial glands. Indeed, the bronchial glands are not of necessity involved.

The Acute Affections of the Lungs accompanied by Dulness on Percussion.

The acute diseases of the lungs are bronchitis, pneumonia, pleurisy, and acute tuberculosis. They have some signs and many symptoms in common. They all present fever; they are all associated with more or less dyspnœa and thoracic pain; they all occasion a cough. The symptoms and signs of acute bronchitis have been discussed. It has been pointed out that the want of intensity of the fever, and particularly the unimpaired resonance on percussion, separate bronchial inflammation from all affections that occasion consolidation or compression of the lung-tissue. We may then proceed to examine the other acute pulmonary affections.

Acute Tuberculosis.—When tuberculosis runs its course rapidly, it is known as acute, tuberculosis, acute phthisis, or galloping consumption. This formidable complaint is met with at the close of other diseases, especially of fevers; but exposure, toil, and anxiety are also among its predisposing causes.

The disorder often begins with a severe chill: fever follows; at first like any fever with anorexia, quickened pulse, and elevated temperature, but soon accompanied by exhausting night-sweats and rapid emaciation, which, in connection with the intense restlessness and prostration, the high temperature, and the supervention of delirium, may cause the febrile disturbance closely to resemble typhoid fever. The symptoms that point to the thoracic malady are the accelerated breathing, the cough, the copious expectoration, the pain in the chest, and the spitting up of florid blood.

The physical signs are not always the same. If the tubercles be scattered through the lungs, no signs are perceived but those of diffused *acute bronchitis;* indeed, the sputum is of the same kind, and tubercle bacilli are not found,[1] or are infrequent. More commonly the signs are like those of chronic pulmonary tuberculosis, and associated with the fever and prostration we find the percussion dulness of a deposit, or the evidences of the destruction of the pulmonary tissue, furnished by coarse moist râles, and cavernous breathing. Tubercle bacilli are then usual.

When the malady assumes the form resembling chronic pulmonary consumption, the diagnosis from bronchitis is not perplexing; but when its phenomena are similar to those of acute bronchitis, the recognition of the tubercular affection may be impossible. This

[1] Von Jacksch, Klinische Diagnostik.

remark applies particularly to the distinction of the miliary form, *acute miliary tuberculosis*, from bronchitis of the finer tubes. From this the diagnosis can be effected only by taking into account that repeated chills, rapid emaciation, and profuse sweats are wanting in the bronchial affection; that the temperature is not so high, nor so irregular; that the râles are more abundant and more perceptible at the lower part of the chest; and that, perhaps, the breathing is not so hurried or so difficult. Moreover, with the intense dyspnœa there are generally frequent and violent fits of coughing, and marked chest pains, in the acute tubercular malady. Yet none of these signs are convincing proofs. The presence of dulness on percussion, or the sinking in at the upper part of the chest, the occurrence of hemorrhage, the finding of the tubercle bacillus, if present, the eruption of miliary tubercles in other organs, and the longer duration of the case are alone conclusive evidence in favor of the acute tubercular disease. Hemorrhage is, however, by no means so constant in the acute as in the chronic form of the affection.

Much the same symptoms will enable us to distinguish between acute tuberculosis of the miliary form and *broncho-pneumonia*, except that we can draw no inference from the dulness on percussion, further than that its early occurrence, with the bronchial symptoms, points to the pneumonic malady; its later occurrence, after the grave symptoms, to the tubercular.

When the dulness on percussion is marked, acute tuberculosis may be mistaken for ordinary pneumonia. But the signs of deposit and of softening in both lungs, and the seat of the lesions at the apices, show differences from a disease which, in the large majority of instances, is one-sided and at the lower part of the lung, which exhibits a characteristic sputum, and in which breaking up of the pulmonary tissue is so rare.

Yet there are cases of acute phthisis that display symptoms and signs very puzzling, and strongly simulating those of pneumonia.

A person in perfectly good health is seized, after exposure, with cough and fever. They are accompanied by dyspnœa, and soon we find signs of consolidation of the lower lobe, or of one lung. The dulness on percussion does not disappear under treatment; and a hollow, blowing respiration and gurgling, usually first perceptible at the angle of the scapula, gradually appear, and indicate the formation of a cavity. Emaciation, which began from the onset, progresses more rapidly, and goes hand in hand with extreme prostration and profuse perspirations. The sputa are copious and purulent, but at no time mixed with blood. The other lung is normal. The case remains

in this condition for several weeks, the patient temporarily improving under stimulants, yet, on the whole, growing weaker and tormented with fever of very irregular type. A slight roughening of the inspiratory murmur, or dry râles at the apex of the unaffected lung, attract attention, and dulness on percussion and the signs of deposition become there more and more manifest. A post-mortem examination exhibits nearly the whole of one lung converted into a uniform yellowish or grayish mass of tubercle, and containing one or several large excavations; not a vestige of healthy lung-structure is to be seen. Scattered tubercles are found in the other lung, and mainly at its apex.

The case just described is one of a group which every physician has met with. The beginning of the case as one of pneumonia or broncho-pneumonia, the persistent consolidation, the occurrence of râles and of subsequent dulness on percussion at the upper part of the previously unaffected side, the continuance of the disease, and the prostration and sweats which accompany it, permit us to foretell its nature and the probable fatal termination, even without the positive evidence of tubercle bacilli in the sputum.

Such cases were not long since classed as *acute pneumonic phthisis*, and looked upon as inflammatory, with resulting caseous infiltration, and its disintegration. With our present knowledge of the bacillar origin of consumption, they are explained by supposing that the tubercle bacilli have fastened readily on the altered lung, or that they have occasioned the attending inflammatory process.

Acute phthisis may simulate other affections besides those of the chest. It has at times the delirium and prostration, the dry tongue, and the bronchial râles of *typhoid fever*. The diarrhœa and the abdominal symptoms are, however, wanting. Yet simultaneous deposition of tubercles in the intestine may cause these; and in this case the chief mark of difference from typhoid fever is the absence of an eruption. Besides, the Widal test is negative, and the thermometric record shows great and sudden variations, to the extent, perhaps, of six or seven degrees, bearing no relation to the number of respirations or to the beats of the pulse. In the blood there is great decrease in the leucocytes, with a relative increase in the polynuclear cells. As there is also a decided diminution of the leucocytes in typhoid fever, but little importance can be attached to the blood-examination in the diagnosis between typhoid fever and acute tuberculosis.[1] Acute tuberculosis lacks the eye-phenomena, the gastric

[1] Warthin, Medical News, Jan. 1896.

disturbance, the rigid muscles, the convulsions, of *meningitis;* else the active delirium it occasionally produces might be attributed to inflammation of the membranes of the brain.

Acute tuberculosis sometimes progresses with extreme rapidity. I have seen a case terminate in thirteen days. It is almost invariably fatal. Yet it has its periods of deceptive improvement: the disease may proceed speedily towards softening, and then remain for a time stationary. In some instances the termination is the result of complications, as of tubercular meningitis, or of erysipelas of the throat and the bronchial tubes.

Acute Pneumonia.—Inflammation of the lung, or "croupous pneumonia," is, in its symptoms, the type of the acute pulmonary affections. The hot, dry skin, the flushed face, the quickened pulse, the extremely rapid breathing, the thoracic pain, the cough, and the peculiar expectoration, point out at once the acute nature of the attack and the organ that is disturbed. Beginning commonly with a chill, or with flushes of heat, the disease progresses with the symptoms indicated.

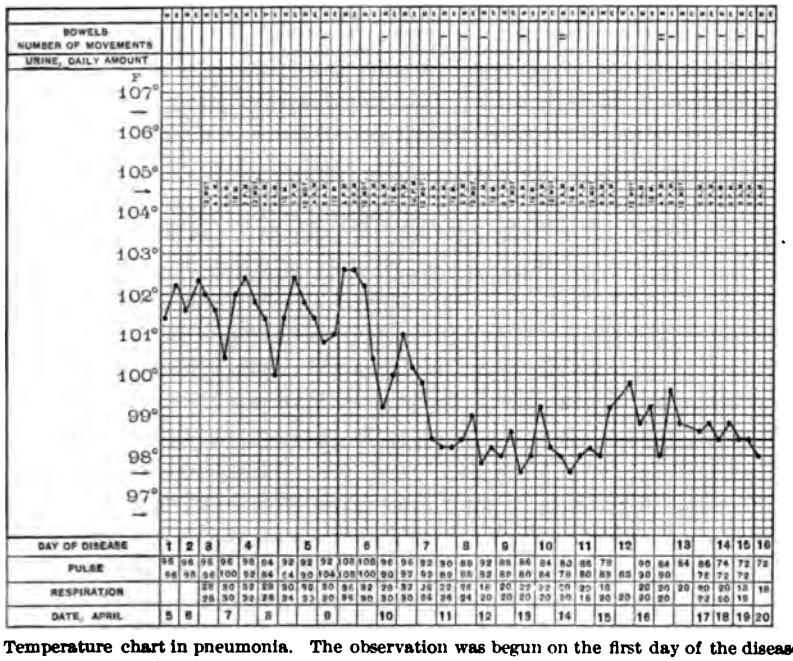
The expectoration is characteristic. It consists at first of a glairy mucus; soon it becomes more viscid, and acquires the appearance dependent upon the admixture of blood with the mucus and exudation matter, to which the term rusty-colored has been given. This rusty sputum is pathognomonic of pneumonia; yet cases run their course without it. The expectoration is sometimes like prune-juice, or it is purulent. Both augur badly: both indicate that destruction of the lung-tissue has begun.

The shortness, or increased frequency, of breathing is another marked symptom. The patient draws from forty to eighty breaths a minute; but the pulse, although rapid, does not quicken in proportion. Pneumonia, therefore, forms an exception to the rule that with greater frequency of breathing the pulse rises. This perverted *pulse respiration-ratio* may be made an important element in the diagnosis. The febrile symptoms are ordinarily severe; still, they are not associated with decided cerebral disturbance. Headache is common; delirium is rare, and, when it occurs, is indicative of danger. In drunkards it may take the form of delirium tremens. The flush on the cheek is so decided that by this and the hurried breathing alone the disease may often be recognized. The flush is generally most obvious when the inflammation affects the apex of the lung. Herpes is also a common symptom.

The temperature rises abruptly, and on the first or second day attains 103° to 105° F. In children and in robust adults it is specially

high. It shows little change, except an evening exacerbation and a marked morning remission of from 1.5° to 2.5° for five to nine days. Between these days, sometimes on the fifth, generally on the seventh day, it falls abruptly, and a true crisis occurs. The temperature may sink to the norm, or even below it, and then another, though not marked, rise take place. At times there happens on the fifth day a partial but decided drop, soon again followed by ascending temperature. This pseudo-crisis is apt to occur in cases that become pro-

FIG. 35.



Temperature chart in pneumonia. The observation was begun on the first day of the disease. The crisis commenced towards the end of the fifth day, and continued through the sixth to the seventh, with a secondary rise on the sixth. The chart is typical, except that the fever temperature throughout was about a degree lower than is usual. There was a slight right-sided pleurisy, but no attending bronchitis.

longed. It is, too, in this class of cases with slow resolution that a gradual termination of the fever is often observed. Sometimes the course of the fever is marked by sudden elevations and striking remissions. This is more common in double than in single pneumonia, and seems to correspond with fresh invasions of lung-tissue.

The urine is high-colored, and that of fever. Nitrate of silver does not precipitate its chlorides. They commonly disappear during consolidation of the lung, and their reappearance shadows forth returning health. The vanishing of the chlorides from the urine hap-

pens also in other acute affections; but in pneumonia it is most absolute.

Pneumonia often exists in combination with other maladies. We find it in association with meningitis, and we must therefore always examine any cerebral symptoms with care; we note it in connection with endocarditis, which may coexist with meningitis; while its association with pleurisy is so common that this can be hardly looked upon as a complication. Among the rarer symptoms are jaundice, parotitis, croupous colitis, milk leg, and transitory aphasia, appearing on the second or the third day.

The *physical signs* vary with the effects of the inflammation. In the first stage, or that of engorgement and beginning exudation in the air-cells, there is only a slight impairment of the normal resonance on percussion. The vesicular murmur is at first somewhat altered; it may be feebler or harsher. But soon are heard with each act of inspiration, and limited to the inspiration, numerous rapidly evolved, very fine, crackling sounds, the "crepitant" or vesicular râles.

As the exudation becomes firmer, and the tissue of the lung solidifies by occlusion of the air-cells, all the signs of complete consolidation are discerned. We find in this stage of red hepatization decided dulness on percussion, unchanged by full inspiration; blowing respiration in its purity, high-pitched and tubular-sounding; bronchophony; and increased vocal fremitus. Râles from the accompanying bronchitis are heard with extreme distinctness through the solidified tissue; so are the sounds of the heart. A crepitant râle is still here and there perceptible, or the ear catches a pleural friction-sound.

When the exudation is reabsorbed or expectorated, the signs of consolidation become less and less perfect. A vesiculo-bronchial succeeds to the bronchial breathing. The dulness on percussion lessens; crepitant râles—not, however, so fine as at the onset of the affection, and mixed with larger moist râles—return; the cough increases; the expectoration becomes more copious, loses its tenacity and rusty color; the dyspnœa diminishes,—all phenomena indicative of the breaking up of the exudation, and of the return of air into the vesicles. If, instead, the exudation be converted extensively into pus, and the lungs soften, the physical signs are the same as in the second stage. The rarity of excavations of sufficient size explains why gurgling and the signs of a cavity are not perceived. We suspect the mischief that is going on within the chest from the protracted dyspnœa, the increasing rapidity of pulse, the purulent or brownish sputa, the pinched features, the dry tongue, and the mental wandering. Recovery may take place even then. This third stage is indeed not so much an abrupt, sud-

denly established process, as it is the extension and greater diffusion of a state that may be found in portions of the lung which to the eye have still all the appearance of red hepatization. It is often impossible to determine that the stage of purulent infiltration or gray hepati-

FIG. 36.

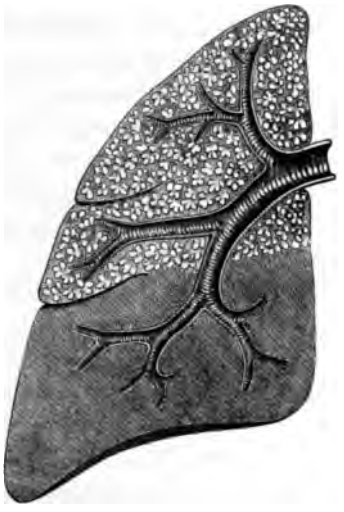


Diagram illustrative of perfect pulmonary consolidation, such as happens in the second stage of pneumonia.

zation has arrived; and death may take place long before the lung presents the condition which pathologists term gray hepatization. We may suspect, from the symptoms, that the pulmonary tissue is seriously damaged. But we can never know it, unless we find the physical signs of extensive softening; and this we very rarely do. True abscess of the lung is extremely infrequent.

The morbid phenomena, physical signs and symptoms of the malady correspond, then, usually in this manner:

PNEUMONIA.

I. Stage of engorgement and beginning exudation.	Crepitant râle; slight percussion dulness.	Cough; beginning dyspnœa and rapidly developed fever.
II. Stage of solidification of lung-tissue (red hepatization).	Percussion dulness; bronchial respiration; bronchophony; often a pleural friction-sound.	Rusty-colored sputum; dyspnœa; cough; temperature generally above 103°, with decided evening exacerbations and morning remissions.

III. Stage of softening (gray hepatization).	The same physical signs as in the second stage; unless large abscesses have formed.	Chills, prostration, purulent or brownish sputum; generally high temperature, 104° to 105°, or upward.

Here is a disease which presents such striking symptoms and signs in nearly all its phases, in which the sputa are so peculiar, the physical signs so distinct, that error is difficult. It becomes still more so, if a few of the pathological peculiarities of pneumonia be borne in mind: the fact that it is rarely double; that it comparatively seldom affects the upper lobe of the lung, and that it is generally accompanied by the signs of pleurisy or of bronchitis. In some instances sudden disturbance of the circulation takes place with the rapid development of cyanosis. These symptoms bespeak a heart-clot, or an acute dilatation of the right side of the heart. Delayed resolution is most often encountered in apex pneumonia.

Let us now contrast pneumonia with the various diseases of the lungs with which it may be confounded. In its first stage, on account of similar signs, the acute inflammatory disorder is sometimes mistaken for œdema of the lung, or for the pulmonary engorgement in some fevers, or for other kinds of congestion of the lungs, and still more frequently these morbid states are mistaken for it.

Pulmonary Œdema.—This consists in the transudation of serum into the air-vesicles. It may be acute, the result of sudden congestion, such as that following injuries of the brain or irritation of the par vagum; or it may arise at the termination of acute affections of the lungs. It is more usually, however, chronic, and is seen as a dropsy of the air-cells, associated with dropsies elsewhere, and in connection with organic disease of the liver, heart, or kidneys. The characteristic manifestations of œdema—be it acute or chronic—are embarrassed breathing, expectoration of frothy serum, and crepitating and fine bubbling sounds diffused over both lungs, and dependent upon the fluid in the air-cells and small bronchial tubes. It presents, thus, many points of similarity to the first stage of acute pneumonia. The dyspnœa, the crepitation in the lung, may well mislead; but we cannot err, if the frothy sputum, the general distribution of the râles, their somewhat coarser character, the bluish lip, the noisy breathing, and the absence of fever be taken into account. In acute œdema these signs are but the precursors of death. In chronic œdema the râles are persistent, and so is the great difficulty in respiration.

Pulmonary Engorgement in Fevers.—In fever of low type a crepitant râle, which might be supposed to be a proof of beginning inflam-

mation of the lung, is often heard at the back part of the chest. The sound is the consequence of pulmonary congestion, with probably slight effusion into the finest bronchial tubes and air-vesicles. It is perceived over both lungs; and this, taken in connection with the history of the case, with the absence of decided shortness of breath, and with the râle not being followed by dulness on percussion and blowing respiration, shows that it is not dependent on inflammation of the pulmonary tissue.

Pulmonary Congestion.—Besides the lung congestion just referred to as occurring in fevers, we have other causes producing a marked congestion, or "hypostatic pneumonia." We find it in enfeebled hearts and in mitral and tricuspid disease, in those whose blood is impoverished and who are for any length of time bedridden, in instances of acute rheumatism, and due to the pressure of tumors. In the dependent portions of the lungs the manifestations of congestion show themselves first; they are, besides the signs of impeded circulation and of deficient aëration of blood, slight expectoration, scarcely any fever, varying shortness of breath, somewhat impaired resonance on percussion at the lower part of the chest,—generally more over the right than over the left lung,—feebleness of respiratory murmur, and a few fine and coarse moist râles. The sputum contains numerous epithelial cells, and blood pigment in various stages of change.

The congestion in all the instances mentioned is passive, and either hypostatic or mechanical. An active congestion of the lungs is a rare condition, though it may come on after strenuous exertion, during mountain climbing, or as subsequent to extreme heat or cold. The physical signs are the same as those of passive congestion; the sputum is apt to contain more blood. There is little, if any fever; and the history of the case, the stationary character of the physical signs, and their double-sidedness, distinguish the congestive disorder from pneumonia.

In its second stage, owing to the cough and dyspnœa, and in part, also, to some similarity in the physical signs, acute pneumonia may be confounded with pulmonary apoplexy, acute pleurisy, acute phthisis, and acute bronchitis.

Pulmonary Apoplexy.—An effusion of blood into the texture of the lung is generally, although by no means invariably, accompanied by external hemorrhage and by great difficulty of breathing. Over the effused blood there is dulness on percussion, and the ear hears an enfeebled or bronchial respiration. Around the seat of the mishap it encounters moist râles. Now, here are signs bearing some resemblance to those of pneumonia. But we miss from among them the

decided fever. We note, on the other hand, not blood intimately mixed with the expectoration, but pure blood, florid or sooty-looking, almost devoid of air, not in large amount, at times surrounded with muco-purulent matter, and ordinarily voided for a number of days. On close scrutiny a grave disease of the heart is generally detected. Then we frequently find the branch of the pulmonary artery leading to the infarcted part plugged by an embolus, which has been formed in the right cavities of the heart or been washed in through the general venous system, and commonly affects the right lung. Again, we have more pain than in pneumonia, and the dyspnœa is different. In pneumonia it augments up to the height of the malady. In pulmonary apoplexy it is greatest, and it is very great, when the blood is extravasated; after that it declines. Yet the two affections often coexist. The closure of the vessel produces a pneumonia from embolism, or the blood acts as a foreign body, and around it is lighted up an inflammation of the lung-structure, which is apt to have its seat in the posterior part of the lower lobe of the right lung; further, the inflammation may be the starting-point of caseous degeneration; or sloughing or gangrene may result.

Pneumonia from embolism may be also caused by a pyæmic condition, and the clots may have their origin in bedsores, in ulcers, and in various forms of suppuration. The plugs are saturated with ichor, and metastatic abscesses supervene. The symptoms are the same, and we can make a diagnosis only by the history; there are the same circumscribed spots of consolidation, and the same kind of pain, which is also often found to be associated with a localized pleurisy, sometimes followed by effusion.

Pulmonary apoplexy is met with in connection with other than thoracic affections. Observations by Brown-Séquard and by Ollivier have proved its association with central nervous lesions, and have demonstrated its occurrence on the same side as the brain-lesion;[1] which is not the case with reference to the ordinary acute pulmonary diseases, for these Rosenbach has shown to be much more frequent on the paralyzed side of the body, and therefore, generally, on the side opposite to the cerebral mischief. Pulmonary apoplexy, or "hemorrhagic infarct," is also met with in malignant fevers.

Of the other diseases mentioned which resemble pneumonia, the distinguishing points need not be here fully described. *Acute pleurisy* will be farther on more particularly studied. With regard to *acute phthisis*, it is only necessary to repeat that cases are encountered,

[1] Arch. Gén. de Méd., Aug. 1873.

apparently of pneumonia, in which, after the symptoms of acute inflammation of the lung pass off, those of phthisis come into the foreground. With reference to *acute bronchitis*, I shall merely recall that no percussion dulness is yielded by an inflamed bronchial membrane. Percussion is thus of signal value in the diagnosis of pneumonia. In fact, when bronchitis complicates pneumonia, and loud, dry râles take the place of the blowing respiration, it is our only trustworthy guide. A single tap on the chest which elicits an absolutely dull sound tells the difference between pure bronchitis and the inflammation of the bronchial mucous membrane which accompanies inflammation of the parenchymatous structure of the lung.

The form of pneumonia most liable to be mistaken for bronchitis is the pneumonia of childhood or of old age, *broncho-pneumonia* or catarrhal pneumonia. But the disease may also occur in adults of any age.

Broncho-Pneumonia.—It mostly supervenes upon acute bronchitis, except in instances in which it arises from inhaling irritating gases. The spread of the disease to the lung-texture is attended with rapid rise of temperature. When the disorder attacks adults, it is apt to seize upon those debilitated by previous disease; it much more commonly affects the upper lobes than does acute croupous pneumonia, and is generally bilateral. As the broncho-pneumonia merely solidifies lobules, the signs of marked consolidation are wanting, or are perceptible over only a small space. Crepitation is not common, but small moist râles are; bronchial breathing and increased fremitus show only over limited points; and the sputum is not rusty and viscid, but catarrhal. Cough and expectoration, sometimes absent in croupous pneumonia, are always present in broncho-pneumonia.

Catarrhal pneumonia, or broncho-pneumonia, is often noticed as a complication of the infectious fevers, especially measles and diphtheria. It is the form of pneumonia developed when particles of food pass into the larynx and bronchial tubes,—aspiration or deglutition pneumonia. Catarrhal pneumonia pursues a much slower course than croupous pneumonia, and generally yields only gradually. The consolidation may continue stationary for weeks, showing a fever with marked daily remissions and exacerbations, like a hectic fever, and then slowly disappear. As interstitial inflammation of the bronchi and alveolar walls is distinctive of the disease, and as the perivesicular structures are markedly involved, persistent local consolidation from interstitial pneumonia or fibroid phthisis often follows. On the other hand, caseous degeneration and breaking-down of the lung-texture may follow, or extended tubercular infiltration become manifest.

Whether the bacillus finds in the consolidated lung a ready lodging, or the broncho-pneumonia is originally excited by the bacillus, phthisis is, in truth, in adults a not uncommon termination; in children, too, this may happen, or rhachitis may develop, or an ill-defined but persistent cachexia, with a great tendency to catch cold.

There is a form of broncho-pneumonia, described as *tuberculous aspiration broncho-pneumonia*, that follows hemorrhage from tubercular cavities. It is usually preceded by active physical effort, and its first manifestation is a hemorrhage.[1] An *aspiration pneumonia* may also follow hæmoptysis from other causes, or be met with as the consequence of aspirated particles from a bronchiectatic cavity, or from an empyema that has ruptured into the lung, or after tracheotomy, or in cancerous affections of the larynx and œsophagus. It not unusually leads to suppuration.

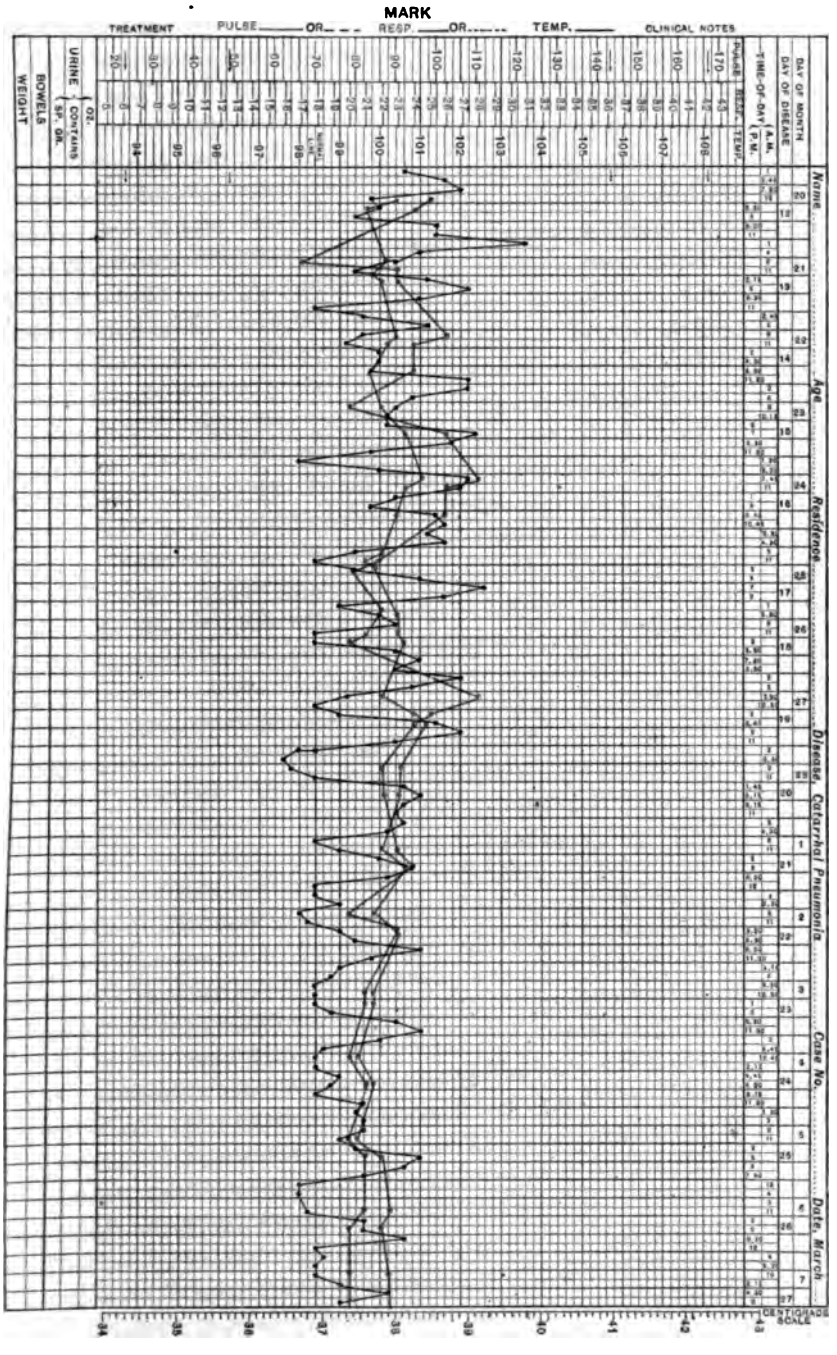
Pneumonia often shows itself in an epidemic form, and is now generally looked upon as an infectious disease, a lung fever; indeed, except as a matter of clinical convenience, it should not be described with pulmonary diseases. The evidence of a micro-organism as its cause is very strong. The diplococcus pneumoniæ was found independently by Pasteur and by Sternberg, and has been fully studied by Fraenkel, after whom it has been named. It is present in the buccal secretion of a certain number of healthy persons. Its association with catarrhal pneumonia is not so close as with croupous pneumonia. In truth, the bacillus of tubercle at times excites this, making a specific broncho-pneumonia from the start; the staphylococcus and the streptococcus pyogenes may also induce it, as Northrup's observations clearly prove.

The cocci are best stained in dilute alcoholic solutions of the aniline dyes, and are readily seen in preparations colored by Gram's method. In this respect they differ from the pneumo-bacillus of Friedlaender, which is also found in a certain proportion of pneumonic lungs, but does not retain the stain after going through the process. The Fraenkel coccus is elongated or round, enveloped in a capsule, and often found in pairs.

The micro-organism of pneumonia generally appears at the height of the malady. It has been found in the blood, in the meningitis that at times attends pneumonia, in the accompanying pleurisy, and in the lung complication of ulcerative endocarditis. The organism is also met with in other conditions than in connection with pneumonia, as in pericarditis, peritonitis, acute synovitis.

[1] Bäumler, Deutsche Medicinische Wochenschrift, No. 1, 1893.

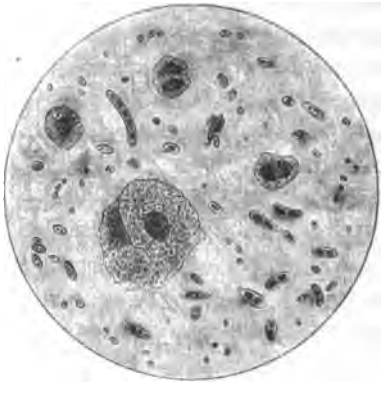
FIG. 37.



Case of broncho-pneumonia lasting six weeks, ending in recovery. The marked evening and morning changes are distinctly seen, and gradually become less and less. The chart shows eight records daily; the part here shown begins with the twelfth day of the disease, and is very characteristic. It is impossible to reproduce here the whole chart. The black line marks the temperature, the red line graphically represents the pulse, the blue line the [illegible]

There are some varieties of pneumonia that present clinical features of a peculiar kind. *Apex pneumonia* is one. It is more usual in children than in adults, and the frequency with which cerebral symptoms arise and draw away attention from the chest is a matter of common observation. The cases, as a rule, are severe, and the temperature is high. *Double pneumonia* differs in nothing from ordinary pneumonia except in the severity of the symptoms. The cases, unless speedily fatal, are generally of longer duration, and the temperature is less characteristic, for the reason that it rarely happens that both lungs are affected at the same time. Double pneumonia is rare; what is called double pneumonia is generally inflammation of one lung and heavy congestion of the other. *Latent pneumonia* is

FIG. 38.



The diplococcus pneumoniæ of Fraenkel; the cocci are stained dark blue, the capsules are unstained. (After von Jaksch.)

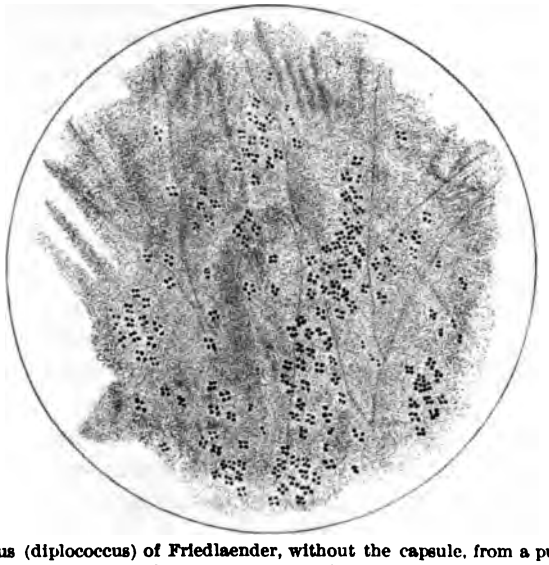
not often seen except in the aged. There is but little fever, and it is only by the physical signs that the disease can be recognized. *Migratory pneumonia*, a condition in which different parts of the lung are successively involved, is not a frequent disease. The temperature shows a tendency to sudden falls, with rapid rises whenever a fresh part of the lung is involved. Some of the older clinicians, especially Wunderlich and Trousseau, regard the disease as having a close connection with erysipelas.

It is always very important to find out whether pneumonia is primary or intercurrent in some other malady, such as in rheumatism, Bright's disease, diabetes, the exanthemata, influenza, the typh-fevers, or in septic states. At times it is distinctly noticed to follow contusions of the chest. As has been already said, it may be epidemic.

By the symptoms and physical signs we cannot distinguish the sporadic and simple cases from those of the infectious malady. Further bacteriological research may solve the matter.

There are two other forms of pneumonia which, as they present somewhat peculiar symptoms, require further to be noticed. They are typhoid pneumonia and bilious pneumonia.

FIG. 39.



Pneumococcus (diplococcus) of Friedlaender, without the capsule, from a pure culture *upon gelatin* from the sputum in a case of croupous pneumonia at the Pennsylvania Hospital. (Drawn by Dr. Joseph Leidy, Jr.)

Typhoid Pneumonia.—The term typhoid pneumonia is applied by some to the inflammation of the lung which may complicate typhus or typhoid fever; it has been also made to include an idiopathic fever in which the affection of the respiratory organs is occasionally wanting. To neither of these maladies rightly belongs the name typhoid pneumonia, since in both the inflammation of the lung is but an incidental accompaniment. Then under the name of pneumo-typhus a disease has been of late years described, especially by German clinicians, in which typhoid fever begins with a well-defined pneumonia, that for the time being throws the enteric symptoms into the shade.

Typhoid pneumonia is pneumonia with symptoms of a typhoid type, and marked by rapid failure of the vital powers. The malady is noticed as a consequence of phlebitis; as supervening in cases of erysipelas, of Bright's disease, and of delirium tremens; or as the sole apparent affection. It happens not infrequently in epidemics,

and is very often observed among negroes. Its ravages on the plantations of South Carolina and Georgia are sometimes frightful. It is, also, very fatal in jails, and among troops in the field serving under unfavorable hygienic conditions.

The physical signs are those of the sthenic form of the disease, except, perhaps, that the crepitant râle is less frequent. Most of the same symptoms, too, show themselves: cough, short breathing, and pain in the chest. All of these may be very marked, or so trifling as hardly to direct attention to the lungs. There is, however, one symptom characteristic and constant, and but one, and that is the great tendency to sinking. As regards the expectoration, it may be rusty-colored; yet occasionally, even in the early stages, it consists of pure blood. The pulse is quick, but weak; dark sordes often collect on the teeth and gums. Pain is absent in some cases, and extremely acute and of a radiating character in others. Concerning delirium. we know that it is much more common than it is in the sthenic variety of pulmonary inflammation, except this affect the apex in children. The flush on the face is usually of a dusky hue, but not invariably: a pink-colored blush, extending sometimes all over the body, has specially attracted attention. The disease is always dangerous, and, as Stokes[1] points out, resolution is extremely slow. Chronic hepatization, with or without a low hectic fever, or a lurking congestion, may continue for weeks.

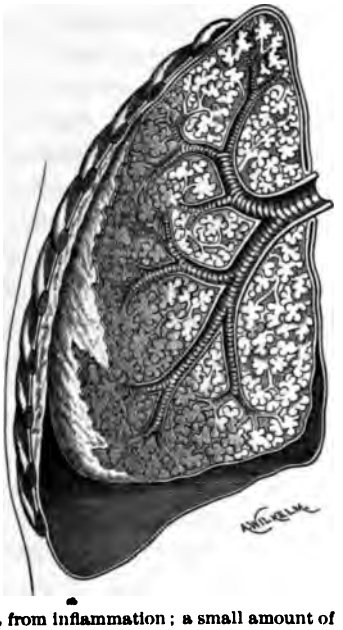
The symptoms of typhoid pneumonia are at times strangely mixed with those produced by other conditions. In many districts in which the complaint is prevalent, it bears the distinct impress of malaria. Again, articular symptoms seem to predominate in some regions of country, and in some epidemics. Gibbes[2] speaks of an acute pain in the back part of the eye, in the ears, or in the side of the neck, attended with stiffness of the muscles; and of a swelling of the tonsils, and of the submaxillary and sublingual glands.

Bilious Pneumonia.—Jaundice and other indications of hepatic and gastric derangement are not usual in ordinary sthenic pneumonia. They may be occasionally caused by the inflammation spreading to the liver, or may be of blood origin. But in the pneumonia so general in the spring and the autumn in the miasmatic regions of some of the Southern and Western States of this country, hepatic symptoms are common, and mark a special type of the disease, known as malarial pneumonia or bilious pneumonia, or by the familiar name of "bilious pleurisy."

[1] Diseases of the Chest. [2] Amer. Journ. Med. Sci., 1842.

This form of inflammation of the lung is simply pneumonia, sthenic or asthenic, on whose features the stamp of malaria is imprinted. The chill with which it begins is usually protracted, and is followed by pain in the side, by fever, by hurried breathing, and by cough. The pain in the side, which depends upon accompanying pleurisy, is sharp, and renders the respiration irregular. The sputum is at times rusty-colored, while at others a frothy and bloody serum or pure blood is expectorated. The fever is much more paroxysmal than in the other varieties of the malady. This peculiarity, and the obvious symptoms of hepatic and gastric disorder, are indeed the only distinguishing traits of bilious pneumonia. The febrile exacerbations are stated by Manson, of North Carolina, to be preceded, during the

FIG. 40.



Roughening of the pleura from inflammation; a small amount of fluid has begun to collect.

morning hours, by an insensible chill,—a coolness of the ends of the nose, fingers, and toes, which, in grave cases, extends over the entire extremities. The rusty sputum has been noticed to occur intermittently in undoubted diplococcus pneumonia.[1] In cases of malarial pneumonia the malarial parasite has been found.

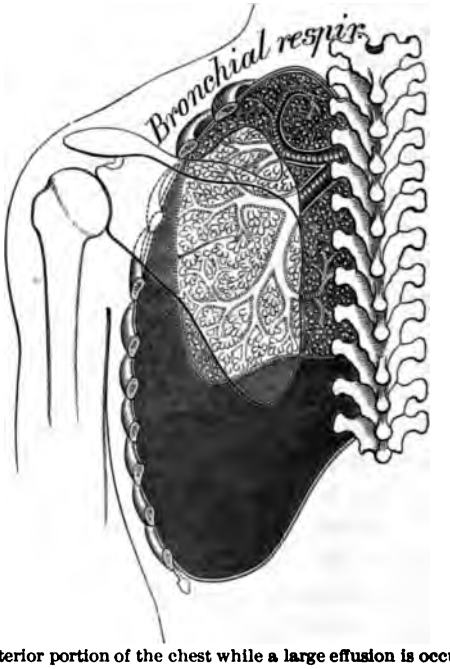
The physical signs are those of ordinary acute pneumonia. Bronchial breathing and bronchophony are said to be more often absent,

[1] Mader, Wiener klinische Wochenschrift, viii. 22, 1895.

or to appear and disappear rapidly. It is certain, if this be true, that in these instances the malady could not have been inflammation, but was more probably a collapse of the pulmonary tissue occurring in the course of malarial fever.

Acute Pleurisy.—Acute pleurisy has been so often incidentally mentioned that a description of its main points will here suffice. It comes on from cold or exposure, or from injuries to the chest; but a great many cases are secondary to some general or infectious malady.

FIG. 41.



Examination of the posterior portion of the chest while a large effusion is occupying the left pleural cavity.

The first effect of the inflammation is to redden the pleural membrane; an exudation of a soft, grayish, easily detached lymph then takes place. This constitutes the first or dry stage of the disease; and if the two inflamed surfaces unite, the disorder does not pass beyond this stage. Often, however, along with the exudation of lymph occurs an effusion of serum, which produces a special train of phenomena, and gives rise to the second stage, or that of liquid effusion.

The physical signs of the *dry* stage are impaired movement of the chest, a feebler respiration, and a friction sound of varying extent and

intensity. The first two signs are caused by the patient instinctively refraining from expanding the lung, because of the pain it occasions. The mechanism of the friction sound, its nature, its superficial character and want of uniformity, have been pointed out in a previous part of this chapter. In the stage of *effusion* the physical signs differ according to the amount of fluid the pleural cavity contains. A moderate quantity of liquid only constricts the lung-texture, and leaves the bronchial tubes intact; a large accumulation compresses everything; it drives all air out of the lung, pushes it into a small space against the vertebral column, and displaces the liver or heart. Wherever the fluid accumulates there is dulness on percussion. When the patient is in the erect posture, the flat sound on striking the chest and the sense of resistance to the finger are marked at the lower part of the thorax, since the fluid naturally settles there. The line of dulness is, however, not the same in front as it is behind. It is generally much higher behind, and alters, of course, with the changing quantity of effusion, and somewhat with the position of the patient. When he lies upon his face, the fluid gravitates, if not circumscribed by adhesions, towards the anterior chest walls, and the percussion dulness posteriorly becomes far less perceptible. The peculiar curve of the percussion line often found has been specially described by Calvin Ellis, and is named by Garland the letter S curve.[1] Another sign of a pleuritic effusion is the one found by Kellock.[2] It consists in percussing posteriorly with force on the ribs of the side suspected with the right hand, while the left hand is placed firmly on the lower part of the thoracic wall just below the nipple. The vibration of the rib struck posteriorly is felt by the left hand in front with greatly increased distinctness, if fluid be present in the pleura.

Where the effusion is extensive, the intercostal spaces are widened and their depressions effaced. The side is distended, fluctuation may be perceived, and, owing to the absolute compression of the lung, no sound is heard over the chest when the patient breathes, or speaks, or coughs. In more moderate collections of fluid, the cessation of sound is not so absolute. There is an ill-defined, deep-seated respiration, and the voice reaches the ear with tolerable distinctness, and occasionally with a peculiar bleating resonance attending it. But, as large collections of fluid are more common than small ones, the former set of phenomena are, at the height of the disease, more frequent than the latter. Occasionally the expiration has a metallic sound, and there

[1] Pneumono-Dynamics, 1878, and New York Medical Journal, Nov. 1879.

[2] Lancet, March 28, 1896.

are resonant râles suggesting a cavity. These pseudo-cavernous signs are most apt to be met with in children.

Above the liquid there is increased resonance on percussion, or a tympanitic sound, Skoda's sound. This tympanitic sound is more manifest at the upper part of the chest in front; it may be, indeed, found in front when it does not exist behind. In some cases the sound has an amphoric, in others a cracked-metal, character. When the ear is applied above the line of percussion dulness, it recognizes occasionally a friction sound; and near the spinal column posteriorly, where the compressed lung lies, it perceives almost invariably distinct bronchial respiration and bronchophony.

When the fluid begins to be absorbed, the voice becomes more audible over the seat of the effusion, the vocal vibrations may be felt by the fingers, and the respiration is again heard. But for a long time it continues enfeebled, and its character is indeterminate; it is neither vesicular nor purely bronchial. As more and more of the fluid disappears, the voice becomes more and more distinct; a friction sound finally shows that the roughened surfaces have come in contact; and the dulness on percussion is replaced by a far clearer sound. False membranes now unite the two pluræ; the intercostal spaces resume their normal shape; and the chest is either restored to its natural size, or is left somewhat contracted. The bronchial breathing near the vertebral column persists for a long time.

These physical signs have been discussed first because they are the most important elements in the diagnosis of pleurisy. The symptoms, indeed, often hardly attract attention; and if we trusted to them, we should be groping in the dark. Pleurisy mostly begins with a chill, followed by fever and by a dry, irritating cough. The most distinctive, though not a constant, symptom of the first stage is the sharp, acute pain, the "stitch in the side." It is commonly felt under the nipple or in the axilla, and is somewhat increased on pressure. Its seat by no means always corresponds to the seat of the friction sound. As the effusion takes place, the pain disappears, dyspnœa becomes evident, and the patient ordinarily lies on the affected side. The febrile symptoms and dry cough continue; yet neither is marked, and both disappear long before the fluid is entirely absorbed. The decubitus is generally on the affected side.

Pleurisy may be idiopathic, coming on generally after exposure to cold and damp; or it may be an attendant upon other diseases of the lungs, such as pneumonia or tuberculosis, or may accompany measles, scarlatina, typhoid and typhus fevers. It may also be caused by wounds of the thoracic walls, by rheumatism, gout, Bright's dis-

ease, diphtheria, pyæmia, cirrhosis of the liver, and other morbid states. We may, too, though rarely, meet with a primary acute tuberculosis of the pleura, which may rapidly become suppurative.

The malady with which acute pleurisy is most likely to be confounded is *acute pneumonia*. Both are affections occasioning dyspnœa; both are, in the majority of cases, one-sided; both present dulness on percussion. But the dulness in the latter disease is far less absolute than in the former; nor do we, save in rare instances, meet with a tympanitic or an amphoric percussion sound in pneumonia, while in pleurisy, as we have just seen, it is far from unusual above the level of the fluid. In the few cases in which a tympanitic or an amphoric sound is perceived in pneumonia, the peculiar tone is most obvious over the consolidated tissue.

The other physical signs of the two diseases show still less similitude. The absence of respiration, of vocal resonance, and of thrill is in striking contrast with the loud blowing respiration, the strong chest-voice, and the increased vocal thrill of pneumonia. There are, however, exceptional cases of pleuritic effusion, in which bronchial breathing is heard all over one side of the chest. Especially does this happen if pneumonic consolidation accompany the effusion; but even in simple compression of the lung, and where the collection of liquid is not extensive, bronchial respiration may be perceived. The difficulty of distinguishing from pneumonia such cases of pleurisy, in which probably the lung-tissue is compressed around the bronchial tubes, is great. As aids in diagnosis, we seek for dilatation of the chest; we note the peculiarities of the breathing, which, although blowing, is mostly fainter than, and unlike, the high-pitched, brazen respiration of pneumonia; we find that the percussion dulness over the upper part and where the bronchial respiration is most distinct is not very great, and, especially, that it disappears on respiratory percussion; we observe that the voice is less strong and ringing, and has, perhaps, a bleating tone; and we take into account the appearance of the sputum and the character of the fever. On the other hand, pneumonia may present itself in a form almost undistinguishable from pleurisy in the stage of effusion; it is when the bronchial tubes as well as the lung structure are filled with a fibrinous exudation. In this *massive pneumonia* we do not find either tubular breathing or fremitus attending the flat percussion note, and it is only by noting the absence of displacement of the heart or the liver, the violent coughing spells, and observing the fragments of moulds of the bronchi in the expectoration that a conclusion can be arrived at.

In the first stage of pleurisy the pain might cause the disease to be

confounded with pleurodynia or intercostal neuralgia. In all three pain is the prominent symptom. Let us see how it differs in each:

Pleurodynia.—Pleurodynia is described as a form of muscular rheumatism. But frequently it is myalgia, or a pleurisy which does not pass beyond the dry stage. Of this nature are most of the fugitive chest-pains from which phthisical patients suffer. Yet there are cases in which no signs whatever of pleurisy exist, but which are attended with as much pain as pleurisy. The pain of pleurodynia is often, indeed, excessively severe; the patient refrains from deep breathing, since every motion of the chest increases his suffering. The pain is augmented by movements of the arm and by pressure, and is generally associated with tenderness. Pleurodynia shares with pleurisy the feeble respiration and the want of action of the affected side. It differs from it by the absence of friction sound and of fever; by the shifting pain, often double-sided; and by the greater tenderness of the chest walls.

Intercostal Neuralgia.—In anæmic women and in consumptives acute thoracic pain is not uncommonly the result of an intercostal neuralgia. The same want of expansion of the chest and the same enfeebled breathing as in pleurodynia are here noted, also the same absence of fever and of pleural friction. The distinguishing marks of intercostal neuralgia are: its intermittent character; its frequent association with uterine disturbance, especially with leucorrhœa, and the limitation of the tenderness to special points in the course of the affected nerve. Valleix has drawn attention to three painful spots which are tender to the touch: one at the exit of the nerve from the spinal column, the second in the axillary region, and the third near the sternum or in the epigastric region. It is on the left side that we are most apt to find intercostal neuralgia, and between the sixth and ninth ribs that the painful places are usually detected.

Pain occurs also in diseases affecting the lung-texture. There is pain of a dull nature in pneumonia, of a more severe character in cancer. But the pain is so dissimilar, and the coexisting symptoms are so unlike, that the confounding of these maladies with pleurisy, on account of the pain, is not likely.

Diseases presenting Dilatation of the Chest, Displacement of the Liver or Heart, and Dyspnœa.

A group of diseases may be here studied, all of which occasion more or less dilatation and prominence of the chest, and all of which are attended with decided shortness of breath. In the recognition of emphysema, pneumothorax, and pleuritic effusion, the dilatation of

the thorax forms one of the main elements; moreover, it is often combined with marked dyspnœa and with displacement of the liver or heart. These affections, then, may be examined in the same connection, and compared with one another, and incidentally with several less common diseases which present similar manifestations.

The history and signs of emphysema were given when treating of the diseases accompanied by clearness on percussion. It was then mentioned that in many instances the prominence of the chest is circumscribed. Such cases cannot be mistaken: the bulging is too limited. But when the emphysema is more general, and an entire side of the chest or the whole chest becomes dilated, or when the inflated lung displaces the liver or heart, the affection comes into the group under consideration. A patient seeks advice for shortness of breath. His chest is inspected, and looks enlarged. The physical signs prove that the disease is not one of the heart or an aneurism. What, then, is it? Is it an effusion into the pleura? is it an intrathoracic tumor? is it pneumothorax? is it emphysema? A tap on the chest goes far towards showing whether it is the former. If the sound rendered be resonant, it is not liquid in the chest that is producing the disturbance, nor, except under rare circumstances, an intrathoracic tumor: the disorder is either pneumothorax or emphysema.

Pneumothorax.—Of all thoracic maladies, pneumothorax is the one the similarity of which to extensive dilatation of the air-cells is the greatest. In both, the large quantity of air occasions increased clearness on percussion; in both, there is considerable and persistent difficulty of breathing; in both, the distention of the chest and the displacement of organs may be obvious. In pneumothorax, however, the symptoms and signs are associated with different conditions. Pneumothorax is an accumulation of air in the pleural cavity, but it is something more: the entrance of air is soon followed by the effusion of liquid.

Air is let into the cavity of the chest by the pleura being perforated by wounds, or through the diaphragm by malignant disease of the stomach or the colon, or, as is most common, by its partial destruction consequent upon disease of the lung. It is in this way pneumothorax originates in the course of tubercular softening, of gangrene, of pneumonia, or from the bursting of a distended air-vesicle or of a dilated bronchial tube.[1] In the large majority of instances it occurs in tubercular patients.

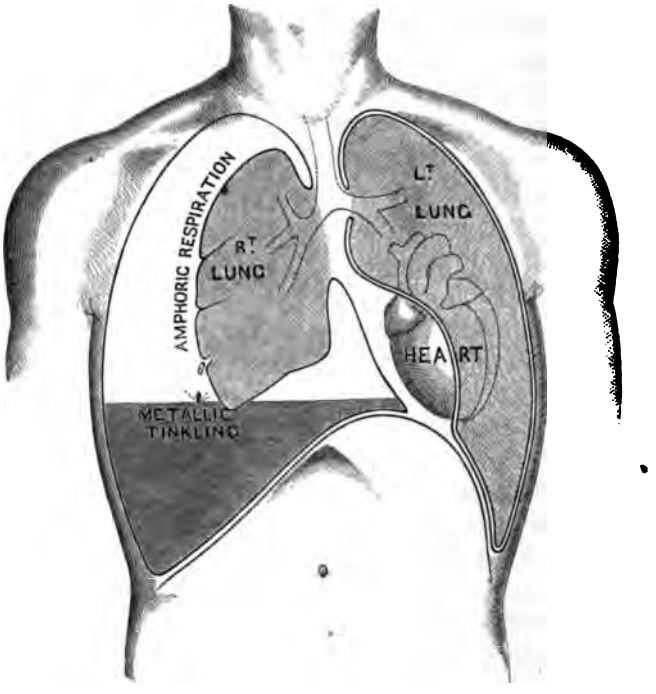
When air passes from the lung into the pleura, it usually happens

[1] Case recorded by Taylor, Prov. Med. Journ., vol. i., 1842.

during a paroxysm of coughing. The pain which ensues is most intense; and with it there is suddenly developed dyspnœa. If death do not take place, symptoms of pleurisy with effusion manifest themselves; and, as in pleurisy, the patient lies ordinarily, but not invariably, on the affected side.

The distinctive marks of pneumothorax are furnished by its physical signs. The ingress of air into the pleural cavity widens the chest,

FIG. 42.



Physical signs in pneumothorax on the right side. The heart is observed to be displaced towards the left, as actually happened in the case from which the outline was taken. The percussion resonance on the right side was tympanitic, extending somewhat over the left margin of the sternum; the fremitus was annulled; the voice metallic.

effaces the depression of the intercostal spaces, and occasions an extremely clear, or, more correctly speaking, a tympanitic, sound on percussion. The air prevents the lung from expanding: hence there is an enfeebled or absent respiration, except near the spinal column, where the compressed organ lies, and where the breathing is bronchial. The hand, if laid on any other portion of the chest, feels, when the patient speaks, no thrill, and no vocal vibration is detected by the ear. When the perforation has not closed, and the air rushes into the artificial cavity produced by the separation of the two sur-

faces of the pleura, the respiration is amphoric, or it, the voice, and the râles are all accompanied by a distinct metallic ring; respiratory percussion, too, changes the sound elicited, rendering it duller. Drops of fluid falling into the cavity, or the bursting of bubbles on the surface of the liquid in the pleura, are echoed to the ear with a metallic sound, and are often heard as a silvery tinkle. A metallic echoing sound is also obtained if the ear be placed on the back over the affected side while a coin is tapped on another coin in front.

The presence of fluid in the pleural cavity gives rise to a dull sound on percussion at the lower part of the chest, which changes readily with the position of the patient, and to a splash, perceptible to the ear and to the finger, when the thorax is suddenly shaken. This continues until the effusion increases, and until the opening closes, the air disappears, and the case resolves itself into one of chronic pleurisy,—the most favorable termination of pneumothorax.

Now let us compare the physical signs with those produced by emphysema. The sound on percussion in both is very clear, or is tympanitic; more so, however, in pneumothorax, which, in addition, exhibits dulness at the lower part of the chest. The respiration in both is feeble. But it is feebler in pneumothorax, and not accompanied by a long, laborious expiration; besides, it is often amphoric, and attended with metallic voice and tinkling,—phenomena which dilated air-cells cannot occasion. Moreover, there can be no splashing sound in emphysema, and this always exists in pneumothorax, except in those rare instances in which there is no fluid in the pleural cavity; on the other hand, the displacement of the heart is generally much greater in pneumothorax, and the dilatation of the chest more apt to be one-sided. Yet too much stress has been laid on the latter point as a means of distinction; for emphysema may be one-sided, and, on the other hand, pneumothorax may occur on both sides. In some cases we are aided in the discrimination by noticing that bulging is perceptible over the displaced heart, and that a metallic echo follows the cardiac sounds. The physical signs of the two diseases are thus very different; so, too, are many of the symptoms. Difficulty of breathing exists in both. But in emphysema it takes generally the form of asthma; besides, it does not set in suddenly and with intensity, and remain intense. In pneumothorax the patient remembers to have been seized with a pain in his chest, since which period he has been continuously short of breath.

Yet there are exceptions to this: there are cases in which the symptoms occasioned by perforation of the pleura are from the onset so slight as not to attract the least attention. Such cases cannot be

recognized, save by their physical signs. Among these, dilatation of the chest, with the widened intercostal spaces, the displacement of the liver or heart, and the exaggerated and altered resonance on percussion are most valuable in preventing the disease from being confounded with some affections which in other respects give rise to many of the same phenomena. In large cavities, for instance, the respiration and voice may be metallic; metallic tinkling, nay, even succussion, may occur. But the prominent chest, the extremely clear, tympanitic, or metallic sound on percussion, bordered by the line of absolute dulness due to the effusion, are not met with. The history also is different, and the dyspnœa is not so great. The same dissimilarities will prevent us from mistaking for pneumothorax a pneumonia in which the percussion sound over the consolidated lung is tympanitic. And a study of the physical signs, too, will at once enable us to discern whether the difficulty in breathing—though it be suddenly developed, and apparently under circumstances which make the swallowing of a foreign body seem likely—be due to this cause, or to perforation of the pleura and pneumothorax.[1]

There is, however, a morbid condition which exhibits nearly all of the signs and many of the symptoms of pneumothorax, and which, were it more frequent, would be the source of constant errors of diagnosis,—*diaphragmatic hernia.*

Of this rare affection we know but little. Yet, what we do know of it teaches us that a protrusion of the abdominal organs through the diaphragm will generally dilate one side of the chest, compress the lung, displace the heart, and result in dyspnœa; and, as the stomach or intestines are, for the most part, the viscera which find their way into the chest, metallic tinkling and a tympanitic sound on percussion are detected. These are also signs of pneumothorax. There is, indeed, no mode of separating the two diseases, except by attention to the history of the case, by noting that the dyspnœa of the former suddenly appears and as suddenly disappears, that it has often existed from birth, and that the metallic tinkling happens when the patient is not breathing, and is mixed up with the rumbling sound arising in the stomach or intestine.

It has been made a question whether we can distinguish ordinary cases of pneumothorax from these very rare ones which are supposed to occur *without perforation.* Now, even admitting that such really happen, as a sequence, for instance, of decomposition in pleuritic effusions, there are no signs by which we can recognize them with

[1] As in a case of the disease described to me by Dr. Walter F. Atlee.

certainty. It has been claimed that there is no antecedent history of a chronic pulmonary affection, particularly of phthisis, that there is not that suddenly occurring severe pain and extreme dyspnœa, that the sputum and breath are never offensive, that metallic tinkling is absent, or rare and inconstant, and that the amphoric breathing is not so well developed or so clearly defined. If in a case of perforation, however, the opening have closed, the physical signs are the same.

Chronic Pleurisy.—Chronic pleurisy is the third of the group of more usual affections characterized by dilatation of the chest, by displacement of the intrathoracic viscera, and by shortness of breath. It is true that acute pleurisy in the stage of effusion would, strictly speaking, find here a place; but the acute symptoms bring it into another class, with which it has been more conveniently described.

Chronic pleurisy is established if the fluid, after an acute attack, be not absorbed, or if an accumulation of liquid take place gradually, in consequence of subacute inflammation of the pleura. It is also found, especially in its purulent form, in a number of infectious diseases, particularly scarlet fever and typhoid fever. This form is also seen to follow pleuro-pneumonia and perforation of the pleura by softening tubercle. Chronic pleurisy has no constant symptoms, and is often remarkably latent: the patient frequently does not remember to have had acute pleurisy. He is not commonly troubled with much cough, nor is the want of breath so great as might be expected; he is not capable of talking for any length of time, or in a loud voice, but he does not really suffer from dyspnœa. His general health may remain good, and no emaciation occur. In some persons, on the other hand, the loss of flesh, the quickened pulse, the sweats, the paroxysms of hectic fever, are so marked as to produce a close resemblance to the last stages of tubercular consumption; and there are cases with misleading marked vasomotor phenomena, with flushing and sweating of one cheek and dilatation of the pupil.

While the differing symptoms rather hide the pleurisy from detection, the physical signs render it easy of recognition. These signs have been studied in describing the effusion in acute pleurisy. It is only necessary to recall that the most significant are absent respiration and voice, a flat sound on percussion, with a vesiculo-bronchial or a bronchial respiration above the seat of the liquid. The intercostal spaces are strikingly widened; their depressions are effaced. They are, indeed, sometimes convex, and the finger pressed on them detects a distinct fluctuation. During the act of breathing, the diseased side is almost motionless, presenting a strong contrast to the obvious play of the healthy side. The lung which is not disturbed increases in

size. Its murmur is more intense, sometimes harsher; and the percussion sound over it is exceedingly clear. In some cases it becomes emphysematous. The heart or liver is displaced. A lateral curvature of the spinal column is apt to take place, and the shoulder remains fixed and stiff during the respiratory acts. To distinguish whether the fluid is collected in one cavity or in several, in other words, whether unilocular or multilocular, is generally impossible. Jaccoud[1] has, however, called attention to some points which aid in arriving at a conclusion. If we have a zone in the dulness where vocal vibrations are preserved, as at the posterior part of the chest from along the vertebral column towards the sternum, and beyond this zone no vibrations are perceived, we may infer that the effusion is divided by a band of pleural adhesion; if the voice and fremitus be preserved, although weakened, over the whole extent of the dulness,—except in a zone of a few finger-breadths at the lower part of the chest behind,—while no tympanitic sound is elicited under the clavicle, we may conclude that the pleurisy is multilocular. When adhesions to the diaphragm exist, the normal movements during respiration at the epigastrium and hypochondrium are reversed, and at each inspiration a marked depression of the inferior intercostal space is perceptible.

Effusions into the pleural sac may last for a long time, and lead to death by progressive exhaustion; or the patient may recover by the fluid being absorbed, or by its finding a vent through the bronchial tubes or the thoracic walls. But the chest is rarely restored to its former state. The lung was too much compressed, or is still bound down by too firm adhesions, to resume its full function. The walls of the chest sink in around it, and the side is flattened, is duller on percussion, and presents a feebler breathing than the other lung, which remains somewhat enlarged. The heart generally returns to its normal position, but the shoulder on the affected side is apt to show a permanent depression.

Notwithstanding the decided character of the physical signs, chronic pleurisy is frequently overlooked; and we hear of patients whose pleural cavity is filled with pus being pronounced incurable consumptives, because they are emaciating and have hectic fever and clubbed nails; or being treated for disease of the heart, on account of the displacement of that organ, and of dyspnœa and œdema; or being dosed with mercury, for an imaginary disorder of the liver; or being subjected to courses of quinine and arsenic, to check a rebellious ague which the chills and paroxysms of fever at times simulate.

[1] Bulletin de l'Académie de Médecine, 1879.

These physical signs are the same whether the fluid be serum or pus. The character of the fluid produces, indeed, no distinctive changes either in the signs or in the symptoms. We suspect empyema if the emaciation be great and accompanied by decided leucocytosis, high temperature, and hectic fever; but I have known pus in the chest with a temperature scarcely above the norm, and, on the other hand, the accumulation not to be purulent with a temperature of 103°. Baccelli has proposed a new and simple test to determine the character of the fluid, which, on the whole, I believe to be of use. It consists in ascertaining accurately how the voice penetrates, especially the whispered voice. If easily and thoroughly transmitted, the liquid is serous and homogeneous; if with difficulty, it is fibrinous or purulent; if not at all, it is the latter. In cases of doubt I have long been in the habit of using a hypodermic syringe and removing with it enough of the fluid for microscopical examination. In those rare instances in which pulsation is noticed, the fluid is only seldom sero-fibrinous; empyema is the rule, and may or may not be associated with an external pulsating tumor.

The *microscopic* and *bacteriological* examination of the exudation will give us valuable information. In rare instances the fluid consists of fat-globules and of masses of cholesterine.[1] In cases of hemorrhagic pleurisy the hæmoglobinometer will inform us accurately as to the amount of blood in the exudation.[2] We find it, indeed, full of blood and altered blood constituents in hemorrhagic pleurisy, a form which pleurisy may assume in cirrhosis of the liver and low fevers, but which is more frequently found in cancerous, and sometimes in tubercular pleurisy. In the latter disease, contrary to expectation, tubercle bacilli, as we know from the observations of Ehrlich, are often absent. There is a group of cases in which, either in a serous or a purulent exudate, we detect the diplococcus pneumoniæ; here there may or may not have been a preceding pneumonia. Cases in which the diplococcus pneumoniæ is met with are apt to set in with acute symptoms like pneumonia, and are generally of favorable prognosis. In septic pleurisy the streptococcus is found, and especially, as Koplik has shown, the streptococcus pyogenes; staphylococci are also met with. These are much more serious cases, both as to duration and as to recovery. A sterile fluid points to tuberculosis. Pleurisies with the typhoid bacillus are sero-fibrinous and of medium gravity.[3]

[1] Debove, Soc. Méd. des Hôpitaux de Paris, tome xviii., 1881.

[2] Henry, Medical News, April 14, 1888.

[3] Fernet, Bull. et Mém. de la Soc. Méd. des Hôp. de Paris, 1895, p. 145.

Leaving out pulmonary consumption, since the points of difference have been already discussed, the affections with which chronic pleurisy, while the pleura is full of liquid and the chest enlarged, is liable to be confounded, are:

Emphysema and Pneumothorax;
Intrathoracic Tumor;
Enlargement of the Liver;
Enlargement of the Spleen;
Abscess in the Thoracic Walls;
Pericardial Effusion;
Hydrothorax.

Emphysema and Pneumothorax.—These, although such different diseases, are grouped together because they give rise, like chronic pleurisy, to a dilated chest, and to displacement of the liver or heart. But the other signs above pointed out, which indicate the presence of air, are so striking that an error in diagnosis can only be the result of carelessness.

Intrathoracic Tumor.—A tumor within the chest may occasion the same distention of its walls, the same displacement of organs, the same dulness on percussion, and the same absent respiration, as an effusion of liquid into the pleura; yet the signs are not exactly alike. There is no fluctuation in the bulging intercostal spaces; the vocal fremitus is not so constantly abolished; and the level of the dulness is not changed by altering the patient's position. Nor is the flat sound so uniform or so strictly limited as that produced by fluid: amid the dulness may be detected here and there a spot yielding on percussion a clear sound. A tumor in the chest, moreover, presses on the nerves, or bronchial tubes, or great vessels, and thus gives rise to severe pain, and to dyspnœa and signs of interrupted circulation far more evident than those caused by pleuritic effusion. It not infrequently grows into the mediastinum, and then leads to prominence of the sternum, and to dilatation of both sides of the chest. These phenomena are found, whatever be the nature of the morbid growth. As most of the thoracic tumors are cancerous, we are often assisted in our diagnosis by discovering a cancer in other parts of the body, as well as enlarged cervical glands, and by noting the severe pain in the chest, the harassing cough, and the expectoration of blood or of a peculiar jelly-like substance. Yet these evidences, while they aid us in establishing the fact of a new growth in the thoracic cavity, do not by any means determine its situation. We cannot say with certainty whether the abnormal formation is situated exclusively in the lung, or in the pleura, or whether it affects both. When the tumor

occupies the mediastinal spaces, and is not cancerous, it is most likely a sarcoma. Lymphadenomata come next in frequency.[1] In children, sarcoma is a more frequent neoplasm than carcinoma.[2]

In those cases in which an effusion into the pleura complicates an intrathoracic tumor, attention to the history and to the signs of pressure alone apprises us of its presence. Yet both signs and symptoms may simulate so closely those of chronic pleurisy as to render a differential diagnosis impossible. Nay, friction sounds, a stitch in the side, and fever may be produced by a *cancer of the pleura*, and be so rapidly developed as to cause the disease to be regarded as an acute or a subacute inflammation of that membrane. The most certain sign is probably the one mentioned by Trousseau,—namely, that the fluid which is evacuated by paracentesis consists of a bloody serum; yet, though of great significance when present, its absence is not so valuable a test, since Moutard Martin found hemorrhagic effusion in only twelve per cent. of the cases he analyzed. Ehrlich[3] has published seven cases, in three of which he detected special cellular elements in the fluid, and was thus enabled to come to a correct conclusion. In some instances there is no fluid in a greatly-thickened cancerous pleura.[4]

It is at times equally impossible to distinguish a *circumscribed pleurisy* from a tumor in the chest. In those rare cases in which adhesions bind the liquid effusion and encyst it, we observe all the marks of a tumor,—a restricted bulging and percussion dulness, and absent respiration and tactile fremitus, though the latter may be retained over the line of the adhesions. Several cysts may form as the result of successive attacks of pleurisy, and exist in any portion of the chest. The fluid may be collected in the mediastinum, or between the lobes of the lung, or anywhere between the surfaces of the pleural membrane. The purulent contents of the sac sometimes find their way into the bronchial tubes, and are expectorated, or give rise to a distinct fluctuation in the intercostal spaces, and then discharge through the thoracic parietes. In such cases the diagnosis is not difficult. But where these phenomena are not present, the dissimilar history of the case and the absence of pressure symptoms are the only means of distinction from a tumor in the chest. Fortunately, encysted pleurisy is a rare disease; were it frequent, it would be a fruitful source

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[1] Hobart A. Hare, Affections of the Mediastinum, 1889.

[2] Edwards, Archives of Pediatrics, July, 1889.

[3] Charité Annalen, 1882.

[4] Purjesz, Deutsches Archiv für klinische Medicin, Aug. 1883.

of error. The same remark applies to hydatid cysts, which may occasion all the signs of a circumscribed pleurisy. An examination of the fluid obtained by an exploratory puncture, in which echinococci are found, is the only positive test.

Enlargement of the Liver.—An enlarged liver usually descends into the abdominal cavity; yet it may be forced upward as far as the fourth rib, and, by encroaching upon the lung, may give rise to many of the physical signs of a pleural effusion. The surest diagnostic test is, that during full inspiration and expiration the line of dulness descends and ascends; while the flat sound of an effusion is not affected by the play of the lungs. This test will be applicable except where the liver is firmly adherent to the walls of the abdomen. As aids in discriminating between the enlargement of the abdominal organ and the presence of liquid in the chest, it may be mentioned that the heart, if at all displaced, is pushed upward and not towards the side; and that the dulness of an enlarged liver extends higher up anteriorly than posteriorly, while the reverse takes place in a pleuritic effusion. Moreover, the respiration at the lower portion of the lung posteriorly, although enfeebled, is still audible.

Enlargement of the Spleen.—An enlarged spleen is attended with prominence and with dulness on percussion at the lower part of the chest on the left side, and might, therefore, mislead into the idea of a pleuritic effusion. Error is prevented by attention to the fact that the dulness extends also downward, and towards the median line, and is much lower on full, held inspiration. Again, the heart is not laterally displaced, but tilted upward; the respiration is feeble, but not absent; and the vocal vibrations are mostly unimpaired.

Abscess in the Thoracic Walls.—This, too, leads to local tumefaction and fluctuation; but we can ascertain whether a fluctuating tumor in the intercostal spaces communicates with the pleural cavity or not—whether, in other words, it is or is not the result of an effusion which is pointing externally—by watching how pressure and the acts of respiration affect it. For unless the diaphragm has become immovable from the extent of the effusion, a bulging which is in connection with the pleura is diminished during a full inspiration, and becomes more prominent when the diaphragm ascends in expiration. The swelling, moreover, can be made to disappear to some extent by pressure. It is not so with an abscess seated in the walls of the chest. It is not reducible, and it does not recede during inspiration.

Pericardial Effusion.—An effusion into the pericardium induces prominence and increased dulness on percussion over the region of

the heart; an effusion into the pleura, dulness and prominence over the back as well as over the front of the lung. An enormously distended pericardial sac will, however, produce a flat sound posteriorly, and give rise to signs of compression of the lung. But in this case attention to the feeble impulse of the heart and its muffled sounds tell us that fluid has accumulated in the pericardium.

Hydrothorax.—The physical signs are the same as those of an effusion due to inflammation; but as the dropsy results from an organic disease of the liver, heart, or kidneys, the serum collects in both pleural sacs. Now, an effusion caused by an inflammation of the pleura is almost always one-sided. Even where both pleuræ are filled with fluid,—a rare condition, except in tubercular pleurisy,—one is affected before the other. This does not happen in hydrothorax. Thus the double-sided effusion, and its usual association with dropsies in other parts of the body, are matters of much significance. Besides, in forming a diagnosis of hydrothorax we may lay stress on the absence of friction sounds; on the smaller quantity of fluid; on the history of the malady; and especially on the presence of a structural lesion of the liver, kidneys, or heart.

These, then, are the diseases with which chronic pleurisy, when it produces dilatation of the chest, may be confounded. Indeed, in view of the frequency of the operation of aspiration or of paracentesis, it is important to know what affections besides chronic pleurisy may lead to prominence of the chest and to compression of the lung; and tapping of the chest has in itself certain diagnostic bearings which may be here mentioned. One of these is an albuminous expectoration that follows, which may be looked upon as a passing albuminuria due to circulatory disturbances. It is not an unfavorable event; on the contrary, in cases in which it happens, retraction of the thoracic parietes is less likely to occur.[1]

Diseases in which Retraction of the Chest occurs.

Chronic Pleurisy.—We may here continue the description of chronic pleurisy in the stage of absorption, since it is under these circumstances that the most marked retraction of the walls of the chest takes place. This shrinking of the thoracic parietes is not a sudden, but a gradual act, and instances are therefore constantly met with in which the upper part of the chest is flattened and the lower, owing to its still containing fluid, bulges. The contraction of one side of the thorax attains its highest degree when the effusion in the pleura is

[1] Legroux, Arch. Gén. de Méd., Aug. 1873.

discharged through the chest walls, and external fistulous openings are established.

The symptoms in the stage of retraction are those of chronic pleurisy with dilatation of the chest, and present, therefore, the same variableness. But œdema of the affected side, which is sometimes so striking a symptom of chronic pleurisy when the effusion is considerable, is here not noticed. The physical signs alter somewhat, according to the presence or absence of fluid in the pleural sac. When none exists, respiration is heard all over the lung as a feeble inspiration with prolonged expiration, or as an indistinct blowing; and now and then a friction sound may be caught. When the pleura still contains liquid, these signs occur at the upper portion of the chest, and a much more absolute dulness on percussion, an absent voice and vocal fremitus at the lower part denote that fluid has there accumulated. The heart is found either in its normal position or still displaced. The force with which contraction takes place may pull it over to the side on which the shrinking is going on. Wasting of the muscles of the shoulder and sensory changes on the affected side of the chest have been observed as a result of chronic adhesive pleurisy.[1]

Now, it is evident that chronic pleurisy, when leading to retraction of one side of the chest, may be mistaken for affections like pulmonary cancer, tubercle, and chronic consolidation, which also occasion a flattening of the chest walls.

From *cancer* we distinguish it by the absence of the peculiar expectoration and of hemorrhage; by the want of signs of perfect consolidation; and by the dissimilar history. We distinguish it from *tubercle* by the diminution of the chest in the latter not being confined to one side; by the physical signs indicative of deposit and softening of the upper portion of the lungs; by the presence of râles; by the occurrence of hemorrhage; by the greater emaciation; and by the tubercle bacilli in the sputum.

Chronic interstitial pneumonia presents, on the whole, most points of resemblance. But there is this difference: the shrinking of the side in this disease is less marked and is confined to the part involved,—usually the lower lobe of the lung. The retraction is much more general in chronic pleurisy; or where it is partial, it is the upper segment of one side of the chest which is flattened,—the lower is prominent, and sounds very dull on percussion, shows no change on respiratory percussion, and yields the ordinary physical evidence of a fluid. In the former malady the blowing respiration, or the enfeebled inspi-

[1] Thévenet, Lyon Médical, 1894, No. 5.

ration and prolonged expiration, and the distinct voice are heard only over the consolidated lobe; in the other lobes the breathing is plainly vesicular. In chronic pleurisy the same abnormal signs, except perhaps the increased voice, are either manifest over an entire side, or are perceived over the narrowed portion of the chest; and at the lower part the respiration, voice, and fremitus are abolished.

In that form of chronic pulmonary induration attended commonly with dilatation of the bronchial tubes, to which the name of *cirrhosis* of the lung,[1] or *fibroid phthisis*, has been given, the flattening of the affected side is as obvious as it is in pleurisy. In truth, the two disorders bear a strong relation to each other. The increased formation of connective tissue in the pleuritic adhesions passes on into the lung, —occasioning an interstitial pneumonia,—though the fibroid change oftener begins in the lung; as this progresses and the lung shrinks, bronchial dilatations usually follow. We distinguish cirrhosis of the lung by the copious and peculiar sputum; by the râles; by the harsh or bronchial or feeble respiration; by the dulness on percussion with an occasional tympanitic note; by the marked resistance of the chest walls; by the increased vocal resonance; by the narrowing of the intercostal spaces; and by the displaced or undiscernible apex beat. The heart may be drawn over to the diseased side, if this be the right side. When the malady is left-sided, further signs of the complaint are that in the second intercostal space to the left of the sternum a double beat of the pulmonary artery is perceptible. Whichever side is diseased shows the diaphragm greatly displaced upward, and a marked vesicular resonance in a line along the edge of the sternum caused by the overlapping of the healthy lung, and in strong contrast with the line of dulness of the cirrhosed organ.[2] The affection is a chronic one, and unattended with fever or laryngeal symptoms. Loss of flesh and of strength is very gradual, and night-sweats are slight or inconstant. Dilatation, or hypertrophy with dilatation, of the right side of the heart, and dropsy, are not infrequent, and hæmoptysis is still oftener met with. It is a mistake to suppose that it occurs only when tubercles are present, or in what is called the bacillary variety of fibroid phthisis.[3]

The disease has among its causes the inhalation of fine particles, such as of steel, of coal-dust, of iron-dust, of cotton. It may have an obscure beginning, or it may clearly date from an acute pneu-

[1] Corrigan, Dublin Quarterly Journal, vol. xiii.

[2] Nothnagel, Sammlung Klinischer Vorträge, 1874.

[3] Sir Andrew Clark, Lancet, July, 1892.

monia, especially an acute or a subacute broncho-pneumonia, or a plastic pleurisy. It may become complicated with tubercle, and then tubercle bacilli are found in the sputum. The fibroid condition in old tubercular lungs or around cavities is an evidence of a disposition towards healing, a local fibroid change, and is not fibroid phthisis. Pulmonary cirrhosis often proves fatal from an acute affection, a pneumonia or a broncho-pneumonia, of the previously healthy lung. In rare instances it is double.[1] Its association with chronic malaria is especially dwelt on by Laveran.[2]

The connection of pleurisy with the cirrhotic lung has just been mentioned; and, though the origin of interstitial pneumonia from invasion through the pleura is in dispute, I hold the view to be correct. We must, however, not forget that primitive dry, or *plastic*, pleurisy is found also under other circumstances, and may give rise to retraction of the chest. Firm fibrous bands may result from organization in the pleura after a dry pleurisy, or after absorption of the effusion; plastic pleurisy may be of tuberculous origin. It is then usually double-sided. Osler[3] mentions some remarkable vasomotor phenomena when these primitive dry pleurisies affect the apex and probably involve the first thoracic ganglion, such as flushing or sweating of one cheek or dilatation of the pupil.

A *collapsed* state of the lung, resulting from a plug of mucus in the bronchial tubes, may yield the manifestations of chronic pleurisy with partial retraction. No signs distinguish such cases, except the more limited depression; the absence of any disease above the flattened spot; the want of friction sound and of tenderness on pressure; and the rapid disappearance of the physical phenomena after an effort of coughing has removed the obstruction.[4]

Where external *fistulous openings* exist, the shrinking of the side, as already stated, is carried to the highest degree. These fistulæ, whether produced artificially or by nature, may persist for months or years, and keep on discharging offensive, purulent matter. The patient emaciates under this continued drain, yet not so quickly as might be imagined. The cough is not ordinarily accompanied by much expectoration. Every now and then, however, a quantity of fetid, purulent sputum is discharged for days. It is then very likely, as Traube has observed, that the pus has softened part of the pulmo-

[1] McCollom, New York State Medical Association, 1885.

[2] Bull. et Mém. de la Soc. Méd. des Hôp. de Paris, 1894, p. 233.

[3] Practice of Medicine.

[4] An instance of the kind is related by Professor William Pepper the elder in the American Journal of the Medical Sciences for April, 1852.

nary pleura sufficiently to soak through the lung into a bronchial tube. It seems certain that it is not the liquid in the pleura which is being voided through a distinct perforation of the pulmonary tissue, for the physical signs of pneumothorax are absent. The clubbing of the nails is often extremely marked, and may exist to an extent far greater than in phthisis. The nail is rounded and bluish, and the whole end of the finger looks enlarged. This appearance is even more striking than the curve of the nail. The nails and last joints of the toes show the same alteration.

The fistulous opening is situated ordinarily in the intercostal space below the nipple. It may, however, be seated at the back of the chest, and communicate by a tortuous sinus with the intestine and other abdominal viscera. A pleuro-bronchial fistula may form; if the opening pass into the lung, the physical evidences of pneumothorax are present, but the side is still retracted, and striking the chest elicits a mixture of a dull and a tympanitic sound. Where merely an external opening exists, no signs of pneumothorax occur, because no air finds its way into the pleural cavity.

A fistulous opening into the pleura is not difficult of diagnosis. It is easy to establish the fact that the fistula is not simply produced by caries of the rib; for a probe may be run into the chest for two, three, or four inches.

SECTION II.

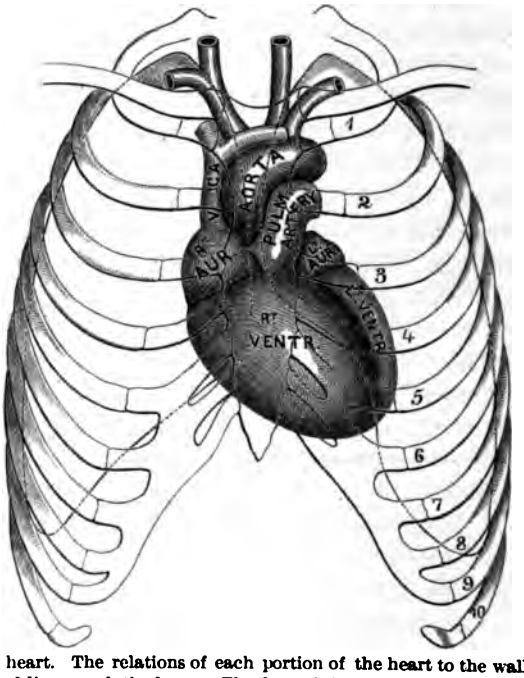
DISEASES OF THE HEART.

The heart is kept from rolling about in the chest by the great vessels which spring from its base, and by the attachment to the diaphragm of its membranous covering,—the pericardium. It lies obliquely in this membrane, with its long axis directed downward and towards the left. Its base points backward and upward towards the right shoulder; its under side rests upon the central tendon of the diaphragm. The valves all lie in close proximity to one another, and within a space of less than an inch square.

The relations the different parts of the organ bear to the chest walls are as follows: The auricles are on a line with the third costal cartilages; the right auricle extends across the sternum to the right of the chest. The right ventricle is placed partly under the sternum, and partly to the left of it. Its inferior border is on a level with the sixth cartilage. The left ventricle lies within the nipple, between the third and fifth intercostal spaces. The apex is seated between the cartilages of the fifth and sixth ribs, to the inner side of, and from an

inch and a half to two inches below, the left nipple. The base of the heart corresponds posteriorly to the sixth and seventh dorsal vertebræ, from which it is separated by the aorta and œsophagus. The greater portion of the anterior surface of the heart is removed from the thoracic walls of the lungs. The right lung extends to the middle of the sternum. The left lung spreads out as far as the fourth cartilage, and covers the whole of the left ventricle, except the apex. The part of the heart which remains exposed consists thus mainly of the lower portion of the right ventricle; it presents the shape of a rough triangle.

FIG. 43.



Topography of the heart. The relations of each portion of the heart to the walls of the chest are shown. The dotted lines mark the lungs. The figure is based upon several careful dissections.

At the left border of the sternum, on a level with the third intercostal space, lies the mitral valve, and in front of this, more directly under the sternum, and but a few lines lower, the tricuspid valve. The pulmonary orifice is seated opposite the junction of the cartilage of the third rib with the left edge of the sternum. Near it, very slightly lower, but placed more obliquely, are the aortic valves. The aorta then proceeds from left to right, and ascends to the upper border on the second costal cartilage on the right side; thence it crosses, under the sternum and in front of the trachea, to the left

side. The pulmonary artery is found in the second intercostal space on the left side, enclosed in the pericardium, and passes to the cartilage of the second rib, where it bifurcates.

The size of the heart is about that of the closed fist. Its mean weight in adults is between eight and nine ounces. Only in very large persons does it exceed this.

The organ exhibits, when in action, a wonderfully perfect mechanism and regularity of movement. Its cavities contract on both sides at the same time, and distend on both sides at the same time. It then rests for a short period. The contraction of the ventricles occasions the impulse which is seen and felt in the fifth intercostal space. While the blood is flowing in and out of the heart, the valves are kept in constant motion. Their play makes itself known by two distinct sounds of unequal length, which are produced mainly by their opening and closing.

The first sound, long and dull, is caused by the forcible closure of the valves at the auriculo-ventricular openings. Yet it is not a purely valvular sound. The stroke of the heart against the walls of the chest, the muscular contraction itself, and the flow of blood into the aorta and the pulmonary artery aid in its formation. The first sound corresponds, therefore, to the closure of the auriculo-ventricular valves, to the impulse of the heart, to the opening of the valves at the orifice of the aorta and of the pulmonary artery, and to the passage of blood along the arteries. The second sound is short, abrupt, and ringing. It results from the sudden closure of the semilunar valves. During its occurrence the blood rushes through the opened mitral and tricuspid valves, and dilates the ventricles.

Examination of the Heart by the Different Methods of Physical Diagnosis.

Before proceeding to examine the heart, we inquire into the history of the case, and into such symptoms as the expression of the face, the appearance of the eye, the condition of the capillary circulation, the presence or absence of dropsical swellings and of cough, the state of the breathing, the character of the pulse, and the frequency and violence of the palpitations. The cardiac region is then explored by the eye and by the hand; the size of the organ is estimated by percussion, and, lastly, its sounds are studied by the stethoscope. These different methods are most conveniently practised when the patient is in an easy position, leaning back in a chair or propped up with pillows in bed. To examine them more in detail:

INSPECTION.

Inspection detects on the chest of some healthy persons a slight protrusion over the seat of the heart; yet this is far from being constant or even the general rule. When the heart is hypertrophied, or when fluid has accumulated in the pericardium, we perceive a marked prominence in the præcordial region. A depression at the lower part of this region may be natural; a very evident depression is almost always the result of an attack of pericardial inflammation.

Yet neither prominence nor depression is a very important sign. One much more so, which inspection shows, is the *impulse* of the heart. This is seen where the apex beats against the walls of the chest: between the fifth and sixth ribs, about an inch inward from the nipple and two inches downward. It is for the most part confined to this point, and appears as a brief raising of the integument, occurring with great regularity of succession. In lean persons it is very distinct; in fat persons it is generally not at all perceptible. Its seat, even in those who are in perfect health, is not always exactly the same. It is changed by different positions, and by the distention of the stomach after a full meal or by flatulence. It is most modified by the acts of respiration. During a long-drawn inspiration the heart descends somewhat and the expanded lung sweeps it inward, and the impulse becomes discernible in the epigastrium. During a fixed expiration the beat moves upward, and appears more extended and weightier. The changes produced in its situation by disease, both thoracic and abdominal, are many. It is tilted upward and outward by the left lobe of an enlarged liver. It is displaced by various affections of the lungs and pleura. It is forced up by a pericardial effusion. It is visible lower down and over a larger surface in enlargements of the heart; but even then it is most distinct at the apex. The apex beat lies without the line of the nipple in most children up to the fourth year.[1]

The alterations in the character and force of the impulse are as diversified as those of its seat. But they are more readily appreciated by the hand than by the eye.

PALPATION.

The extent and force of the beat are changed in a number of cardiac affections, both functional and organic. Both are temporarily increased by powerful excitement; both are permanently augmented

[1] J. Mitchell Bruce, Enlargement of the Heart, in Keating's Cyclopædia of the Diseases of Children, vol. ii.

by hypertrophy. In dilatation and pericardial effusion, the extent over which the stroke is felt is greater than in health; but the impulse is feeble, and in the latter disease irregular and wavy. Softening of the texture of the heart, diseases of the brain, some morbid states of the blood, many infective fevers, and a low condition of the system will also enfeeble the beat.

The hand, when laid on the præcordial region, perceives at times *two* impulses. This double impulse is often recognizable in health, especially in thin persons. It becomes still more evident in hypertrophy with dilatation of the ventricles. One of the beats is systolic; the other corresponds to the diastole. Bouillaud cites examples in which the diastolic stroke was double.

All these modifications of the impulse stand in direct connection with the action of the ventricles. The auricles, save in some rare instances in which they are dilated and their walls thickened, give rise to no perceptible movement in the chest wall.

The sounds of the heart can be analyzed by placing the hand over the cardiac region. They will be felt, the one as a long and dull, the other as a short and distinct, vibration. The motion is due to the play of the valves, and disappears with their destruction. The fingers applied over the heart perceive at times a peculiar thrill, or a rubbing movement. The first—called by Laennec, from its resemblance to the purr of a cat, the purring tremor—is nearly always indicative of a valvular lesion, especially of mitral obstruction. The second is caused by the to-and-fro motion of a roughened pericardium.

PERCUSSION.

Percussion affords the readiest means of judging of the size of the heart. The patient is placed in a recumbent position; then, by a series of moderately strong taps, we proceed downward from near the middle of the left clavicle, until a dull sound, accompanied by decided resistance, tells that we are striking over a solid organ. The point at which this dull sound begins is over, or immediately at the lower border of, the fourth cartilage. It corresponds to the upper limit of the portion of the heart which is left uncovered by the lung.

The superior border of the dulness having been thus ascertained, we next percuss on the right side of the sternum, on about a level with the fifth rib, and progress across the bone. At, or very near to, its left edge we find marked resistance and a duller sound. Here we draw our second line, and continue to strike straight across the cardiac region up to the point at which a clear, full note demonstrates that

the pulmonary tissue is resounding. This determines the transverse diameter of the heart,—at least so far as it can be mapped out on the chest. The apex of the organ and its inferior surface remain to be fixed. The first is readily done by advancing in an oblique direction from the already ascertained right border. But we can save ourselves this trouble by feeling for the impulse or by listening for it with a stethoscope.

The inferior surface can be circumscribed by prolonging the line of the dulness on percussion of the upper border of the liver, and then judging by the greater amount of resistance and the fall in pitch that the heart has been reached. The dulness elicited by percussing the cardiac region is not so absolute as that of the liver or of some other solids. It is mixed with the sound of the lung-tissue, or with the resonance of the sternum. Nor is it a representation of the size of the entire organ. It simply portrays the more superficial portion, which is uncovered by the lungs.

In women it is particularly difficult to define these limits. It can be done only by having the mammary gland drawn to one side while percussing. It is equally difficult in children, as the space over which the dulness is perceived is very small. In adults the dulness ordinarily spreads over two, or nearly two, intercostal spaces. Its transverse diameter in a grown person of medium size is about two inches and a half. In tall, broad-chested men it is upward of three inches. Such, at all events, is the result of measurements I have made.

The range of the dulness is changed by a number of causes, physiological as well as pathological. A full inspiration alters it materially, by bringing the lung down over the heart, and by displacing the organ itself. The upper border of the percussion dulness shifts to the extent of an intercostal space. Below the nipple, between the fifth and sixth ribs, the sound becomes clear; but over the dislodged lower part of the heart, the beat of which is distinctly seen under the cartilages of the ribs, at a point varying from three-fourths of an inch to one and a fourth inches from the median line, there is dulness with resistance to the finger. A full expiration produces converse phenomena. It enlarges the boundaries, especially in an upward and transverse direction. The dulness reaches nearly, or even entirely, across the sternum. Auscultatory percussion enables us to fix the percussion limits more closely. Sansom's pleximeter also conduces to greater acuracy in cardiac percussion;[1] so does the phonendoscope. The absolute size of the heart is best determined

[1] See paper by William Ewart, Lancet, Aug. 1891.

by the Roentgen rays; the fluoroscope makes very clear the changes in size and shape produced by respiratory and other movements.

The area of dulness is diminished in emphysema. It is increased by a shrinking of the left lung, and by diseases of the heart and of its membranes. Prominent among these are hypertrophy, dilatation, and an effusion into the pericardial sac.

AUSCULTATION.

The sounds of the heart are audible at all parts of the præcordial region, but not everywhere with equal distinctness. The first, the systolic sound, being more ventricular in origin, is best heard over the lower part of the heart; the second, the diastolic sound, is valvular and best defined at the base.

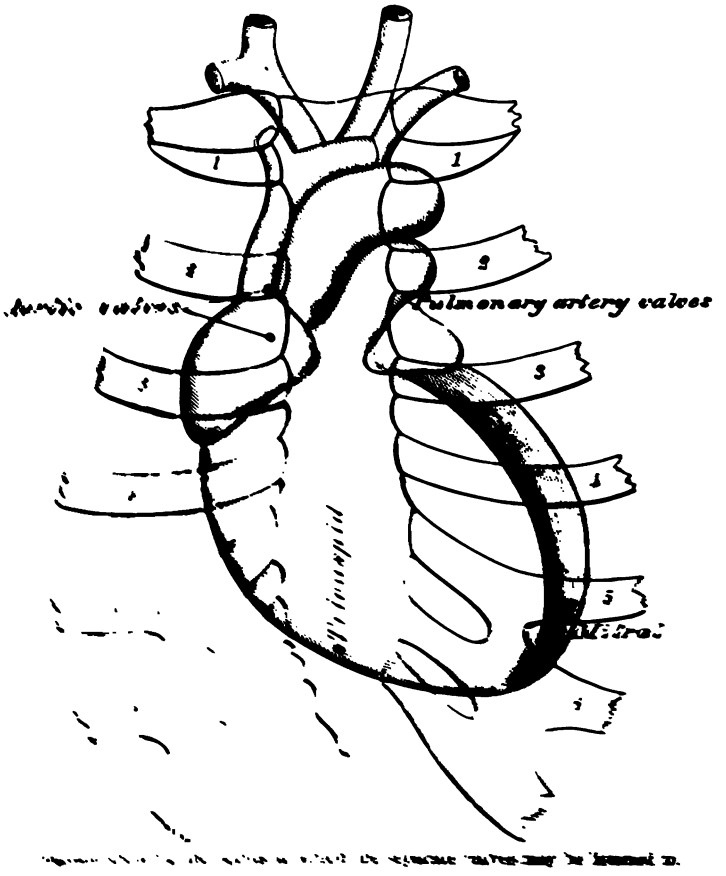
Each of the valves forms a separate sound, or at least a portion of one. Now, experience teaches that there are points at which the sounds of the several parts of the heart may be isolated. Some of these points accord with the anatomical seat of the valves; others do not. None do so very closely; and the proximity of the valves to one another is such as to make it desirable that the localities selected for listening to them should be some distance apart.

Clinical observation sanctions the following: the sounds of the aorta are to be studied at the right edge of the sternum, in the second intercostal space; from there the stethoscope may be carried to the second costal cartilage of the right side, the "aortic cartilage," and down to the left edge of the sternum opposite the third intercostal space,—that is, not far from the seat of the aortic valves. The pulmonary orifice lies very close to them: but the artery itself ascends to the second costal cartilage on the left side. Its sound may be isolated in the second intercostal space, near the left edge of the sternum. The mitral is listened to immediately above the beat of the apex. The sounds of the tricuspid and of the right ventricle may be sought for in the vicinity of and somewhat above the ensiform cartilage.

Both sounds are discerned at each of these points. But the same sound varies in different situations. The first sound over the left ventricle near the apex of the heart is dull, heavy, and prolonged; that over the right ventricle is clearer, shorter, and of higher pitch. The second sound heard there presents no constant and appreciable variance from that over the left ventricle; yet it is less ringing and distinct than the second sound of the pulmonary artery and aorta. Even these two are not precisely alike. The second sound of the latter, when compared with that of the former, is found to be sharper and more accentuated. The first sound, however, does not differ

materially from that of the pulmonary artery. But the first sound of both does differ most materially from that over the ventricles. Compared with the first sound over the right ventricle, the first sound of the pulmonary artery is much duller, more indistinct and like a vibration, and not of so high a pitch. Compared with the first sound at the apex, the first sound of the aorta lacks the weighty, prolonged character which belongs to the ventricular sound.

FIG. 44.



During a full inspiration, the sounds at the interspace between the second and third costal cartilages on the left side disappear almost entirely, and become faint at the aortic cartilage. The first sound at the apex lessens also very much in distinctness, but it is better heard at a new point of impulse, visible towards the median line and just below the cartilages of the ribs. During a full expiration, the extent over which the heart-sounds are perceived is increased.

The sounds grow in loudness in any functional disturbance of the heart. When the organ is palpitating violently under strong nervous excitement, they may become short and sharp, and sometimes so loud and ringing as to be audible to the by-standers. They are often permanently louder than in health, and are shorter and more clearly defined when the walls of the heart are thinned. This is particularly the case with the *first* sound. When the walls of the heart are thick, the first sound over the hypertrophied portion is apt to be dull and prolonged. The first sound is weakened if the structure of the heart be softened: hence it is feeble in some low fevers, and in fatty degeneration of the organ. It is also less distinct when there is a want of tone in the muscle, or when the mitral and tricuspid valves are thickened.

To determine whether a dull first sound at the apex be due to an injured mitral valve, or to an alteration of the muscular power of the heart, Flint advises to place the stethoscope over the apex of the heart, and then on the outside of the left nipple to isolate the element of impulsion, which unites with the valvular element to form the complex first sound. If there be a marked impulse over the apex, but if by means of the stethoscope placed to the left we perceive no sound which possesses a valvular character, or hear a sound only faintly valvular, we infer that the mitral valves are damaged.

The *second* sound is not so liable to be changed as the first. It is rendered somewhat duller by a thickening of the semilunar valves; on the other hand, it is more ringing when they are thin, and in great functional excitement of the heart, and in altered blood conditions, as in lithæmia or in gout. The sound, indeed, always becomes more distinctly accentuated if the column of blood closes the valves forcibly. This occurs not infrequently in hypertrophy of the ventricles, especially the left, and in the increased tension of the vessels in contracted kidney and in arteriosclerosis; it affects the second aortic sound. Accentuation of the second sound also takes place where a decided obstruction exists to the passage of blood through the lungs, and in mitral valvular disease. In the latter conditions it is over the pulmonary artery alone that this accentuated second sound is audible.

Both the sounds are occasionally obscure. This happens when fluid has accumulated in the pericardium. The sounds may be changed in their relative proportion to each other, and the pauses between them be lengthened or shortened, or else the sounds may intermit from time to time. From this perverted rhythm we do not derive any definite instruction as to the condition causing it. It may be associated with organic disease or exist without it. The same may be said of reduplication of the sounds of the heart. The second sound is the one which is generally split. Yet both of them may be doubled, or one may be doubled over one part of the heart and not over another; so that four or three sounds are counted to each beat of the pulse. The cause of the reduplication is the want of synchronous action of the two sides of the heart. The direct value for diagnosis of the altered movement is not great. Yet there is some value to be attached to the changed rhythm. Thus, the peculiar alteration of the sounds, which causes us to hear three sounds during the action of the heart, two of them in the diastole, producing the rhythm that has been likened to the gallop of a horse, is often found in contracted kidney and in arteriosclerosis. It is particularly heard over the mitral and the tricuspid region. Fraentzel[1] has noted the frequent occurrence of this gallop rhythm in typhoid fever and in croupous pneumonia, and looks upon it as a sign of grave cardiac weakness; it is also a sign of serious import in chronic Bright's disease.

Such, then, are the modifications which the healthy sounds present. At times we meet with sounds which do not in the least resemble those naturally heard, and which overshadow them or take their place. They are called *murmurs*, and are mainly produced either within the heart or on its surface.

Those murmurs that are *endocardial* have a common quality: they are more or less blowing. Yet the sound is not always of the same character or pitch. It may be low-toned, it may be high-pitched; it may be soft, it may be harsh; it may resemble the blowing of a bellows; it may be musical; it may be filing, or rasping, or sawing. The ingenuity of every listener exerts itself in tracing a similarity to some familiar noise; but to little practical purpose. These different sounds teach us nothing certain as to their source. They are, moreover, not at all times the same in the same case, since the heart when excited may emit a sound different from that which it does when it is beating quietly.

[1] Krankheiten des Herzens, Berlin, 1889; see also Cuffer and Barbillion, Arch. Gén. de Méd., 1887.

A blowing sound originates in the altered relation of the blood to the part over which it moves. This general statement opens the way to the consideration of the specially acting elements, both in the blood and in the heart itself.

Usually a cardiac murmur springs from a change at one of the orifices. This may be either a narrowing or a roughening, which interposes a local obstruction to the flow of the blood; or it may be an insufficiency to close the opening. In the latter case the blood regurgitates, and a murmur is occasioned by the deviation of the direction of the current and the establishment of another. This subversion of the course of the circulating fluid, added to its increased velocity and force, is the chief source of those temporary blowing sounds not infrequently perceived when a heart is greatly excited, while both its valvular apparatus and its muscular texture are healthy. Obstruction to the circulation, with, perhaps, altered position of the heart, is the cause of the cardiac murmurs in pleurisy and in pneumonia. But we meet often with instances where none of these causes are present, and where altered blood is the foundation of the murmur.

Thus, to sum up the subject, we find murmurs that depend upon organic change, and murmurs that are unconnected with any structural alteration; and these inorganic murmurs are due either to an unnatural condition of the blood or to temporarily perverted action or position of the heart.

The murmurs, however caused, have different effects on the sounds of the heart. They either accompany the sound throughout the whole or a part of its duration, and thus obscure it, or else they take its place and hinder it from being generated. In time of their occurrence they correspond to the contraction or to the dilatation of the heart, and therefore to the first or to the second sound; at least, they do so practically. It is true, they may immediately precede or succeed either sound, or fill mainly the intervals of silence between them, or occur early or late in the sound; but attention to such minute divisions, except in the case of the presystolic murmur, is for most purposes unnecessary. In point of fact, it is often difficult enough to say whether the murmur we hear is systolic or diastolic. The readiest method of judging of the time of the production of a murmur is to feel for the impulse while listening with the stethoscope. The blowing sound which agrees with the beat of the heart is systolic; the one just before the systole is presystolic; the one between the beats is diastolic.

When a murmur is once established it attends each motion of the

heart that can give rise to it; but it is not always equally perceptible. It may become very faint, or disappear entirely, by the patient changing his position. It is sometimes manifest only when the heart is acting strongly. Indeed, it always requires a certain force and velocity in the passage of the blood to generate a murmur. Yet overaction of the heart may be as destructive of its distinctness as diminished action. This is, however, a matter that, should it be desirable for diagnosis, we can control by the administration of medicines like digitalis, aconite, or veratrum viride, provided their use be not contra-indicated.

A murmur is sometimes heard by the patient himself, or is audible before the ear is placed over the heart. It may be perceived as an abrupt blowing sound, apparently coming out of the mouth. I have met with a number of such instances. The murmur is nearly always systolic.

Posture exerts a decided effect upon murmurs. A blowing sound distinct in the recumbent position may become very faint or disappear when the patient stands erect, and the reverse holds good, although less common; anæmic murmurs are thought to be more intense in the recumbent position.[1] Pressure, too, has an influence upon the abnormal cardiac sound; it notably augments it, and often raises its pitch. Yet pressing the stethoscope against the chest does not occasion as much alteration in endocardial as it does in pericardial sounds.

A murmur may be obscured by the respiratory sound; and the natural sounds of the lungs may be mistaken for blowing sounds in the heart. Certainly the resemblance is often great; but blunders may be readily avoided by listening to the heart while the patient suspends his breathing.

Having ascertained positively the existence and the time of occurrence of an endocardial murmur, the next thing is to determine its exact seat, and, if possible, its immediate cause. The *seat* of the murmur is judged of by the place of its greatest intensity, and by the relation this bears to one of the four points for the clinical examination of the heart above described. If it be most distinct at or near the apex of the heart, it is produced at the mitral orifice; if immediately above or at the ensiform cartilage, it is generated in the right ventricle and at the tricuspid opening. If we hear it most plainly at the sternum, somewhat towards its left border on a level with the third intercostal space or even the fourth rib, and with equal or

[1] James H. Hutchinson, Amer. Journ. Med. Sci., April, 1872.

nearly equal distinctness at the second costal cartilage on the right side, we are enabled to decide that it is developed at the origin of the aorta. The pulmonary artery is not often the seat of a murmur. When it is, this is clearly perceptible in the second intercostal space on the left side, and extends, if the valves be diseased, to the junction of the third left cartilage with the sternum; although we must bear in mind that occasionally in mitral affections the murmur is loudest in the pulmonary area, or, as Naunyn has shown, not exactly over the artery, but rather an inch and a half or more from the left edge of the sternum in the second interspace.

Any of these situations may be the site of a distinct murmur occupying only one sound of the heart, or being produced in both,—one murmur taking place with, the other against, the current of blood. Yet it rarely happens that the murmur is strictly limited to one of these positions: it will mostly extend in various directions from its point of intensity, growing fainter and fainter as this is left. A blowing murmur thus transmitted may drown the natural sounds of the heart at the parts not diseased. But when one orifice alone is affected, we can usually hear the sounds at the other valves. They may be obscured, but still they exist; and it is a vast aid when they are heard, since they set the limits of the disease. How important is it, then, to examine each portion of the heart separately, as much for the purpose of saying what is not as what is deranged!

If satisfied as to the seat of the murmur, we naturally turn to inquire into its origin. Is it caused by an alteration of the valves? Is it unconnected with any appreciable change of structure in the heart? There is nothing in the murmur itself which will tell us positively. As a rule, it is true that a harsh murmur results from organic disease, and a soft murmur is inorganic; but we judge with much more certainty by the time of the occurrence of the blowing sound and by the accompanying phenomena. A murmur presystolic or diastolic is organic; a systolic murmur may or may not be organic. A murmur arising from an impoverished state of the blood is systolic, generally soft, of low pitch, is perceived over the base of the heart, and is accompanied by a humming sound in the veins of the neck. It may be heard over the right base, or on the left side over the pulmonary artery; although Balfour maintains that it is not really over the pulmonary artery, but about half an inch or more to the left of the pulmonary area, and is not an arterial, but an auricular sound.

Throughout the consideration of the endocardial murmurs, they have been treated as originating at the seat of the valves. In truth,

it is there that they are formed. Still, they are occasionally due to morbid states in the body of the ventricle, or in the auricle. But in either case this is very rare. As regards the auricles, they yield no appreciable sound in health, nor are they in disease, except rarely, the source either of sound or of murmur.

A blowing sound is not of necessity limited to the heart: it may be transmitted all over the arterial system. Yet it would be a great mistake to suppose that every murmur heard over the arteries is connected with a disease of the heart. It is often but the sign of impoverished blood, or a sound dependent upon local roughening or narrowing of the tube. The latter may be temporarily produced by the pressure of a stethoscope.

Let us now examine the sounds which originate on the outside of the heart. These *pericardial murmurs* have all a common source: they all result from irregularities on the membrane. Like the pleura, the smooth serous covering of the heart moves noiselessly in health; but when it is roughened by a deposit of any kind, the friction of its surface gives rise to a sound which may be single, but which is usually double. The character of this sound is variable. It may be a to-and-fro rubbing murmur, or it may be grazing, or scratching, or creaking, or whistling, or clicking and resembling the valvular sounds. It has but one quality which is constant, and that is its superficiality. By this superficiality; by the strict limitation of the sound to the region of the heart; by its altering from time to time its precise seat; by its greater extent and intensity when the patient bends forward; by its occasional increase, and even change of character, on external pressure; by its following, rather than occurring with, the movements of the heart; and by the sensation of friction which it communicates to the finger,—we know that the sound heard is produced on the surface of the heart. Yet, in spite of this array of points of difference, it is often difficult to distinguish a pericardial from an endocardial murmur.

An error not easy at times to avoid is the failure to discriminate between the presystolic apex murmur, regarded as characteristic of mitral constriction, and a pericardial friction localized near the apex. The only trustworthy points of distinction are that the pericardial sound changes in its quality and loudness; that it is rendered stronger and changed in pitch by pressure exerted with the stethoscope, and that the second sound at the left base is unaltered.

A friction sound is prone to mask the natural sounds of the heart. At times, although heard over the cardiac region, it is not due to inflammation of the pericardium. The exudation may be on the sur-

face of the pleura adjacent to the pericardium, and the murmurs be caused solely by the movements of the heart, with the rhythm of which they coincide. Sometimes, again, the sound heard in the cardiac region is in reality the rubbing of an inflamed pleura. If any doubt exist, let the patient be told to suspend his breathing. As this is stopped, the pleural sound ceases.

Such is a brief description of the different physical signs met with in examining the heart, both in health and in disease. Their importance for diagnosis it is difficult to overestimate. A knowledge of the physical signs is the solid foundation, without which any structure that may be raised will soon tumble to pieces.

The General and Local Symptoms of Diseases of the Heart.

It is not easy to say what are and what are not the symptoms that belong to diseases of the heart. There are vital manifestations directing attention to the heart which are not associated with any change in its structure; and most serious changes in its structure may occur without any of these vital manifestations. Yet we often find a significant group of symptoms that accompany an affection of the heart. Some of these attest directly the organ disturbed, such as pain in the cardiac region and palpitation. Others are the indirect and more remote expressions of its derangement, such as cough, dyspnœa, hemorrhages, dropsy, disorders of the brain and nervous system, engorgement of the abdominal viscera, a peculiar state of the arteries and veins, and the aspect of the face. It is unnecessary to do more than mention some of these, since several have been already described in connection with pulmonary complaints, and there is nothing in the cough or in the shortness of breath by which we can absolutely determine it to be caused by a disease of the heart. The same with respect to hemorrhage; there is nothing characteristic about it. It simply proves the efforts of the blood-vessels to relieve themselves of the strain which the disturbance in the flow of blood has put on them. The capillaries and the smaller blood-vessels give way first; partly from the reason just assigned, and partly from the altered state of their coats, a common associate of cardiac disease. These hemorrhages are prone to happen from the bronchial tubes and the lungs, and the blood is expectorated; but they may also take place directly into the pulmonary tissue, or into or from any part of the body. Their danger is in proportion to the amount, to the importance of the function of the structures into which the blood is effused, and to the possibility of its finding an outlet. The peril is greatest when the blood is poured into the brain.

Cardiac Dropsy.—The dropsy caused by disease of the heart is met with in different situations: in the cellular tissues, in the peritoneal and pleural cavities, in the pericardium, in the ventricles of the brain and under the arachnoid, in the air-cells of the lungs,—in fact, in any part where fluid can exude, and where there is a space which can receive.

In anasarca dependent upon a cardiac lesion, the dropsical swelling begins about the ankles and feet. The accumulation is much influenced by position; the feet are more puffy towards evening, when the patient has been all day in the erect posture, and least so when he gets up in the morning. The dropsy is most constantly found to be associated with disturbance in the flow of the venous blood, and therefore with disorder of the right side of the heart, particularly with a dilatation of the cavities. It may be permanent or not. Its extent certainly does not bear a constant relation to the extent of the cardiac disease. It bears a more constant relation to the amount of venous congestion, and to the impoverishment of the blood.

Derangement of the Circulation.—Unmistakable evidence of the obstruction to the flow of blood through the veins is afforded by their prominence in different portions of the body. This is especially manifest in the superficial veins of the neck, which, moreover, when the tricuspid orifice is permanently open, exhibit a distinct pulsation with each beat of the heart. The turgid condition of the venous system is rendered equally obvious by the livid tinge of the skin and the bluish color of the lip, and by ramifications of fine bluish vessels on the surface. But the arterial system may also be gorged, and we may find the capillaries and the smaller arteries seemingly ready to burst. The conjunctiva is then highly injected, and the cheek has a coarse, red look. This change in the color and appearance of the face, the thickening of the eyelids, and the prominent eye, make up the peculiar physiognomy of a chronic cardiac malady. The state of the larger arteries is very variable, and mainly according to the nature of the disorder and the condition of the cardiac walls and of the blood-pressure. The pulse may be small and tense; it may be full; it may be rebounding; it may be very irregular; and it is often out of all proportion to the forcible action of the heart.

The derangement of the circulation of individual parts manifests itself by special symptoms. It shows itself in the brain by attacks of cerebral congestion; by vertigo; by violent headache, occurring in spells, or, less acute, in dull persistent ache, increased on exertion,—a form especially met with in children. We see evidences of the congestion of the nervous system in the disturbed dreams; in the sudden

starting up from sleep; in the irregular action of certain muscles; in the spots which float before the eye. It is possible that the strange sense of insecurity and the irritability, of which patients afflicted with a cardiac malady complain, are produced by the same cause. At any rate, whether produced thus or not, they are remarkable symptoms. There is no disease which unnerves more than a disease of the heart. Indeed, the mere fear of its presence gives rise to restlessness and gloom, and breeds timidity in those who would look any external danger boldly in the face.

The disordered flow of blood through the abdominal viscera occasions organic changes and a disturbance of the functions of the several organs. Thus, the liver increases in size, or undergoes other alterations which interfere more or less seriously with the elimination of the bile; or the kidneys no longer secrete as in health, but become much engorged and drain off the albumin of the blood; or the spleen sustains textural transformations. These states all tend to give rise to more and more dropsy, and hence to more and more suffering.

The symptoms which point most directly to the heart itself are palpitation and irregularity of action, and pain. These symptoms denote that the function of the organ is disturbed, or that its innervation is in some manner deranged; but they denote nothing more. They are, therefore, common to functional derangement which occurs associated with structural changes in the heart, and to purely functional derangement.

Cardiac Pain.—Pain in or over the heart is met with in both acute and in chronic diseases; yet it is not a regular or well-defined symptom of either. When we reflect that the heart may be pinched, may be torn, without exciting any suffering, it will be readily understood why its disorders do not occasion much pain. Indeed, many a case of enormous enlargement of the heart, or of profound textural alteration of its walls or valvular apparatus, is unaccompanied by pain. Still, we meet with instances in which distress at the heart and various uneasy sensations are among the more marked symptoms of a chronic cardiac lesion; and we even find persons complaining of a persistent pain in the heart, which extends to the left side of the neck and arm, in whom this symptom has preceded the signs of a disease of the heart or of its great vessels. The greatest suffering happens in the obscure malady termed angina pectoris.

Angina Pectoris.—The disease occasions paroxysms of intolerable anguish. These come on suddenly, and pass off as suddenly. Their main feature is an agonizing pain in the præcordia, as if the heart were being firmly grasped by an invisible hand, or as if it were being

torn to pieces. The pain is, however, not limited to the cardiac region; it radiates in various directions, shooting to the back, to the neck, and especially down the left arm. But this is not all: worse than the pain are the intense anxiety and the feeling of impending death. The heart palpitates during the fit. Yet, if we judge by the character of the pulse, its movements are not always materially disturbed; for this may be but little altered, and regular; very generally the arterial tension is high. Again, there may be a decided difference between the pulses, the left being almost or quite imperceptible.[1] The face is generally pale. Difficulty in breathing, contrary to what might be expected, is not a prominent symptom, and is, in fact, often wanting, while sometimes there is asthmatic wheezing, or the breathing is irregular and of the Cheyne-Stokes variety. Giddiness, spasmodic seizures, temporary coma, perverted sensibility, occasionally attend or follow the cardiac attack, and so does pericarditis.[2]

The duration of the fits is as uncertain as are the causes which excite them. They may cease in a few minutes; they may last an hour. They come on rapidly, without any assignable reason, though they are generally produced by exertion, by fatigue, by exposure to cold, or by mental emotion. However provoked, they are always dangerous. The heart may stop beating during the paroxysm. "My life is in the hands of any rascal who chooses to annoy and tease me," was a saying of John Hunter's. And in truth, after he had suffered for years from these seizures, his irascible temper brought on one in which he expired. It happens sometimes that the second attack follows at a short interval the one by which the disease first declares itself, and proves fatal. Latham[3] narrates the history of two cases of this kind. In one, life ceased in a fortnight after the first seizure; in the other, in ten days. Nay, it may be cut short even in the midst of the first manifestation of the malady. Such was the death of Arnold of Rugby.[4] On the other hand, I have had a patient under my care who for weeks at a time has five or six attacks daily, kept in check, but not wholly averted, by nitrite of amyl; and in another patient as many as forty occurred in one day.

The immediate conditions on which the symptoms of the attack depend are veiled in obscurity. Whether they be or be not produced

[1] Hamilton Osgood, Amer. Journ. Med. Sci., Oct. 1875.

[2] Clin. Soc. Transact., vol. xvii. p. 82.

[3] Lectures on Diseases of the Heart, vol. ii.

[4] Stanley, Life and Correspondence of Thomas Arnold.

by temporary increase of weakness in an already enfeebled organ; whether a cardiac spasm occur or do not occur; whether the pain and the sensation of approaching death be or be not caused by an acute distention of the heart with blood,—we do not know. All we do know positively is that the excessive pain abruptly appearing and disappearing points to deranged innervation. Yet we can go a step farther; we can say with certainty that angina pectoris is not often an uncomplicated neuralgia. Modern research has taught us that these outbreaks of a cardiac neurosis are frequently linked to some structural change. This structural change, so far as we can now see, is, however, not at all times the same. The list of disorders of the heart and arteries which angina pectoris may accompany is, indeed, very long. There is hardly an affection of the walls or cavities of the heart, scarcely a morbid condition of the arteries that nourish it or spring from it, with which the distressing malady has not been observed to be associated. It has been found as an attendant on changes in the coronary artery; on every form of valvular disease; on thinning of the parietes of the heart; on adherent pericardium; on fungoid growths springing from the apex of the organ.[1] It has been thought that combined with all of these states is fatty degeneration, which thus would be at the root of the angina.[2] Whether this view be correct or not, it is certain that fatty degeneration is very often conjoined with angina. But fatty degeneration occurs also without angina, as does disease of the coronary arteries, and we are thus forced to admit that, however frequent the association, some unknown element is still the determining cause. Yet arteriosclerosis, general or localized, in the heart or aorta, with changes in the myocardium, is the most common obvious lesion. In influenza and in diabetes angina is also met with. During the attack, as Brunton has shown, there is a vasomotor spasm of the smaller vessels, with a rise in blood-pressure and increased tension in the arteries.

Angina pectoris is now very generally ranked among the vasomotor neuroses. But evidence is not wanting, as Peter's cases prove,[3] that neuritis of the cardiac plexus, the neuritis itself being consecutive to aortitis, is the cause of a certain number of cases.

Angina pectoris is easy of recognition. The points to ascertain are, whether it is linked to an organic cause, and to what organic cause, or whether it is a pure neurosis, either primary or reflected. It

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[1] B. Travers, Medico-Chirurgical Transactions, vol. xvii.

[2] Quain, Medico-Chirurgical Transactions, vol. xxxiii.

[3] La Semaine Médicale, March, 1892.

may be a question whether those *severe pains in the region of the heart*, which occur in feeble anæmic persons after unaccustomed exertion, or which are brought on by the excessive use of tobacco or of tea, or which happen in rheumatic or gouty subjects, especially while suffering from indigestion, are real angina, or whether they may be separated from this affection. They differ from it, irrespective of being far less violent and less radiating, by the circumstances leading to an attack, and by their constant association with palpitation. *Intercostal neuralgia* with palpitation might be mistaken for angina; but the painful spots in the course of the affected nerve, and the comparatively slight suffering, distinguish it. In truth, it is a complaint seated only in the thoracic walls, and referred by the patient to the heart. *Great irritability of the heart*, attended with faintness, with sensations of sinking, with flushing alternating with pallor, and with pain, due most likely to a neurosis of the cardiac plexus, is discriminated from true angina by the palpitations, by their connection with pain which never rises to the anguish of angina pectoris, by the periodical nature of the pain, its nocturnal occurrence, and its duration for one or two hours. Often, too, this apparent or false angina is found in persons who are hysterical, or are subject to neuralgia, or are laboring under a disorder of one of the abdominal viscera, and is then clearly reflex. It must be, however, admitted that the distinction between true and false angina is one of degree rather than of kind.

Another complaint that may be confounded with angina is what may be called *cardiac epilepsy*. In this rare affection intense pain in the region of the heart happens in paroxysms. But unconsciousness, however temporary, occurs also, and the pain is apt to follow rather than to precede the unconsciousness. Yet it may outlast it, and become associated with twitching of the muscles of the face and with other spasmodic movements. These, the unconsciousness, and the time at which the pain happens, distinguish the malady from those instances of angina in which, owing to the severity of the pain, the patient passes into a protracted faint.

Palpitation.—This arises in various affections of the heart, organic as well as functional. It bears no positive relation to any special cardiac malady. So, too, with irregular rhythm of the heart's action, with which palpitation is often combined, and which, when linked to a disease of the organ, generally means failing heart-muscle. But palpitation, with or without irregular rhythm, may take place in a sound heart, disturbed temporarily by the condition of the nervous system, or of the digestive organs, or by toxic influences.

Often the pulsations of the heart become stronger, more extensive,

and more perceptible, from mere nervous excitement. But it is not necessary to detail the symptoms of a purely nervous palpitation. Every one has experienced them. Every one knows that there is a feeling of slight constriction about the chest, with a hurried breathing, and a strange sensation as if the heart were leaping from its place. Every one is also aware that the organ is felt thumping against the walls of the chest, and with a force which shakes them. The popular notion, that the heart is the seat of the emotions, is based on these striking evidences of its disturbed action.

During an attack of palpitation the cardiac sounds are clear and ringing; in neurasthenics and anæmics, or if the cardiac excitement be prolonged and violent, a systolic murmur at the apex or left base is not uncommon.

Persistent rapidity of cardiac action, or *tachycardia*, may happen without obvious cause in persons apparently healthy. It is very common in irritable hearts and in exophthalmic goitre. Spender[1] has called attention to its occurrence among the earlier signs of rheumatoid arthritis. The extreme frequency of the action of the heart is in some instances remarkable. I have known it to beat over two hundred times in the minute. The disorder may occur in paroxysms, described as "cardiac nerve storms" by H. C. Wood.[2] Great rapidity may be joined to a condition in which the two sounds are precisely alike, and the pauses of equal length. This fœtal rhythm, or *embryocardia*, is a sign of heart debility, and is most frequently seen in connection with marked dilatation, or in fevers. In *gallop rhythm* the cardiac sounds are split, most often the second. It is generally found associated with the weakening heart of arteriosclerosis and of interstitial nephritis.

On the other hand, the heart-beat may be very slow, less than thirty times in the minute. We may find this slow action, *bradycardia*, both in functional and in organic maladies, though it is most likely that the nerve-centres are in both affected in the same way.[3] Bradycardia is often associated with atheroma of the aorta or of the coronary arteries. It is also met with in a number of instances of fatty heart and in old-standing valvular disease. Its association with jaundice, with uræmia, with lead poisoning, with feeble heart action during convalescence from fevers, with apoplexy, with epilepsy, with

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[1] On Osteo-Arthritis, London, 1889.

[2] University Medical Magazine, March, 1891.

[3] See an interesting analysis of ninety-one cases, by Prentiss, Transact. Assoc. Amer. Phys., vol. iv., 1889.

affections of the medulla and the cervical cord, and with melancholia, is well known.

FUNCTIONAL DISORDERS OF THE HEART.

It has just been stated that the direct symptoms of a cardiac disorder—pain, palpitation, irregular action—are met with when no recognizable structural change has taken place. Under such circumstances the affection of the heart is termed functional, and its symptoms are those mentioned, variously combined, sometimes the one predominating, sometimes the other. These functional disorders are very much more frequent than the organic. They are, for the most part, produced by direct excitement of the heart, or by its being sympathetically disturbed by a source of irritation away from it, or in the system at large. The symptoms may be said to constitute the disease.

Functional Disorders characterized by Palpitation, associated or not with Change of Rhythm.

We have already briefly mentioned the causes of augmented action which are associated with organic changes, and those which occasion temporary disturbance of the heart. A more lasting form of palpitation is engendered when the organ is kept constantly excited by a deranged condition of some viscus remote from it; by the use of stimulating substances; or by some general morbid state. Thus, a disordered stomach or liver leads to a reflex disturbance of the heart, which ceases if the disorder of the stomach or liver be remedied. In gouty, lithæmic, and rheumatic persons the heart frequently pulsates with increased quickness, and sometimes with marked irregularity. Special articles of diet, especially tea or coffee, produce palpitation; so does the inordinate use of tobacco and of alcohol. Overwork, worry, immoderate dancing, masturbation, and excessive sexual indulgence, but particularly masturbation, are prolific sources of continued palpitation. Women who are hysterical, or whose uterine functions are disordered, suffer from palpitation. So do anæmic persons and neurasthenics complain of the beating of the heart.

A troublesome kind of palpitation is that attended with marked *irregularity* of the action of the heart, displaying itself by the beat being now slow, now fast, or occasionally intermitting. Sufferers from lithæmia or gout, or old persons with feeble digestion, are particularly liable to it. This form of palpitation is not without danger. It is prone to be associated with an alteration in the structure of the heart, such as flabbiness of the walls.

Some who experience fits of palpitation faint away during these. But the almost complete suspension of the movements of the heart which characterizes an attack of syncope has no definite connection with any form of palpitation, nor, indeed, with any form of cardiac disorder, organic or functional. In those who are subject to attacks of palpitation or to irregular action of the heart, the organ may finally become enlarged.

A peculiar irregular action of the heart has been much discussed under the name of *hemisystole*. Leyden describes cases in which with every two beats of the heart only one beat of the pulse is felt, and attributes this to the right ventricle contracting alternately with the left. Different explanations have been given of the fact, but the observations of Riegel and Lachmann, while they do not strictly confirm the alternate action of the ventricles as the cause of the phenomenon, point to irregular contraction of the muscles of the heart as the cause.[1]

We sometimes meet with a singular form of functional disturbance of the heart which leads to textural changes, and to which Graves called particular attention. It consists in a long-continued excitement of the organ, as evidenced by its increased force and rapid and irregular action, which is followed by a swelling of the thyroid gland, pulsation of the arteries of the neck, and prominence of the eyeballs. This disease, *exophthalmic goitre*, is most commonly observed in women, and connected with hysteria, neuralgia, or uterine disturbance; it has in some instances an evident origin in worry or in shock. It is generally considered to be owing to an affection of the cervical sympathetic nerve. But its cause is far from certain. There are those who hold it to be a neurosis of the nerve-centres, especially of the vagus centre; and the detection of ptomaines in the urine is thought to be a proof that this apparent neuro-cardial malady is really consequent upon secondary disturbance of the nervous system due to poisonous products from the thyroid.

The most characteristic sign, the greatly accelerated heart's action, varies much in extent. All the signs may remit or may become aggravated from time to time, and especially during a severe attack of palpitation. The turgescence of the thyroid gland arises quite independently of the usual exciting causes of bronchocele. It is accompanied by a pulsating thrill similar to that of an aneurismal varix, and by a distinct throb. At an advanced period of the complaint, these signs subside, and the gland becomes more solid. In-

[1] Virchow's Archiv, Bd. xliv.; Deutsches Arch. f. klin. Med., Bd. xxvii. p. 393.

deed, the whole affection may disappear, and the gland, the eyes, the beat of the carotids, the action of the heart, all return to a normal condition. On the other hand, hypertrophy and dilatation may result from the cardiac palpitations, or the malady be noticed in association with valvular disease, under circumstances which make it a question whether this has followed it or is a mere concomitant.

The protrusion of the eyeball is often combined with a symptom that Graefe particularly observed,—a want of agreement between the movement of the lid and the raising or depressing of the glance. The palpebral aperture is wide, owing chiefly to spasm of the upper lid, and this spasm of the elevator of the upper eyelid is held to be pathognomonic.[1] Another symptom of importance is trembling of the hands. The tremor is fine, and, as Charcot pointed out, affects the whole hand, but not individual fingers. There is also, as Charcot shows, greatly lessened resistance to the galvanic current; but this sign is not of much value, as Cardew[2] has found the electric resistance to diminish greatly whenever the skin is moist. Other symptoms are cramps, usually at night, epistaxis, œdema of the legs and eyelids, lessened respiratory expansion, moderate elevation of temperature, sensation of heat, flushed and moist skin, paroxysmal attacks of diarrhœa, atony of the large intestine, intermittent swelling and pain in the joints, pigmentation, urticaria, pruritus, bulimia without gain in flesh, emaciation, glycosuria, migraine, rheumatic symptoms, and mental derangement. All the physical manifestations of the disease are double-sided; and this, with the unchanged state of the pupils, serves to distinguish it from those rare cases[3] where a thyroid growth pressing on the sympathetic on one side produces symptoms of exophthalmic goitre, including the palpitations.

In the distinction from ordinary goitre, the absence of eye and heart symptoms is of most value. There is also no murmur heard over the enlarged thyroid gland; whereas in Graves's disease a continuous murmur there is most common, and is, indeed, looked upon by Guttmann as of the greatest diagnostic importance, especially aiding us in those doubtful cases in which the exophthalmos is wanting. My own experience confirms this statement.

There is another form of functional disorder of the heart so peculiar as to demand a special notice. It is the curious cardiac malady

[1] Abadie, La France Médicale, vol. ii., 1881.

[2] Lancet, Feb. 1891.

[3] Eulenberg, Ziemssen's Cyclopædia.

of which we saw so many examples in soldiers during our civil war, to which I gave the name of "*irritable heart*," and which we also find occurring in private life. Its main symptoms are habitual frequency of the action of the heart, constantly recurring attacks of palpitation, and pain referred to the lower portion of the præcordial region. The palpitations come on chiefly during exercise, but may also take place when the patient is quiet, and in many cases happen most often at night, thus interfering with sleep. Those who are subject to the disorder complain much of headache and of dizziness, and especially of being thus affected when suffering from palpitation. The pain is generally dull and constant, but is often also described as shooting, and as taking place only in paroxysms. Its chief seat is near the apex, and it is combined commonly with excessive cutaneous sensibility. Often there is pain nowhere else in the body; but in some instances the cardiac distress is associated with pain in the back, which itself is not unusually connected with the excretion of oxalate of lime by the kidneys.

The action of the heart is very rapid, and in many instances its rhythm is irregular. The impulse is slightly extended, but not forcible, like that of hypertrophy: it is rather abrupt and jerky. As a rule, to which I have met with but few exceptions, the sounds of the heart are modified as follows: the first sound is short, sometimes sharp, resembling the second sound; at other times it is extremely deficient and hardly recognizable; the distinctness of the second sound is much heightened. We either hear no murmurs in the heart or in the neck, or they are inconstant. The area of percussion dulness does not appear to be augmented. The pulse is almost always easily compressible; it may or may not share the character of the impulse. It is usually very much influenced by position, falling rapidly twenty beats or more when the erect posture is exchanged for the recumbent. The increased frequency of beat is not connected with increased frequency of respiration, for often with a pulse of one hundred the respirations scarcely exceed twenty in the minute. The disorder is very obstinate, and improvement comes but slowly.

The cause of the morbid cardiac impressibility is difficult to ascertain. It seems in many instances to have followed fatiguing marches; in some, to have occurred after fevers or diarrhœa; it was not connected with scurvy, or with the abuse of tobacco. That it was not due to anæmia was proved by the general aspect of the men, which was often that of ruddy health. Similar conditions of the heart occur from excessive dancing, excessive smoking, and certain occupations,

such as glass-blowing. For a fuller consideration of the subject I refer to observations elsewhere detailed.[1]

Yet another form of functional cardiac disorder is the one which I have described under the name of *cardiac asthenia*, or heart exhaustion. It shows essentially the signs of a weak heart, and follows long-continued worry and overwork. There is rapidity of cardiac movement with very feeble action, and a great tendency to faintness. The breathing is singularly undisturbed. The impulse of the heart is weak, the first sound short, valvular, the capillary circulation defective. The duration of the cases is a long one, and recovery takes place but gradually. In the cases that are not purely nervous, but in which the heart-muscle is enfeebled, shortness of breath and functional dynamic apex murmurs are often noticed.[2]

These, then, are the principal varieties of functional disorder of the heart. It is hardly necessary again to state that the physical signs present the most certain, if not the only, means of distinguishing the functional from the structural affection. They show us that neither the size of the organ nor its sounds, with the exceptions above mentioned, are materially different from what they are in health.

The irritable heart just described, as indeed other forms of functional heart disorder, may pass into organic cardiac disease by the constant overaction of the heart. And *overaction* or *strain* may also, as I have proved in the publications just referred to, lead to valvular affection, sometimes by preceding hypertrophy, at other times by a slow process of inflammation or disorganization engendered at or near the seat of the valve. Of this I published several instances in the "Memoirs of the Sanitary Commission." Others have been brought forward by Allbutt[3] that happened among persons engaged in vocations requiring sustained and oft-repeated muscular effort,—such as lifters, smiths, sawyers. And in his elaborate monograph, Seitz[4] has detailed several fatal cases in which the symptoms of a fatigued heart, due to strain, were followed by extensive dilatation without valvular disease. Leyden, too, has added to our accurate knowledge of the subject.[5]

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[1] Medical Memoirs of the U. S. Sanitary Commission, 1867; American Journal of the Medical Sciences, January, 1871; and the Third Toner Lecture, Smithsonian Institution, 1874, "On Strain and Overaction of the Heart," where also the forms of irritable heart occurring in civil life are described.

[2] Amer. Journ. Med. Sci., April, 1894.

[3] St. George's Hospital Reports, 1872.

[4] Die Ueberanstrengung des Herzens, 1875.

[5] Die Herzkrankheiten in Folge von Ueberanstrengung, Berlin, 1886.

ORGANIC DISEASES OF THE HEART.

Organic diseases of the heart may be classified as follows:

ORGANIC DISEASES OF THE HEART.

Diseases affecting the walls of the heart, and mostly changing the size of the cavities.		Hypertrophy. Dilatation. Atrophy.
Diseases affecting chiefly the walls alone		Fatty degeneration. Parenchymatous degeneration. Fibroid heart, cardio-sclerosis, etc. Malformations. Rupture of the heart. Injuries and wounds. Aneurism of the heart. New growths and parasites.
Inflammations	of membranes.	Endocarditis. Pericarditis.
	of muscular structure.	Myocarditis (Carditis).
Diseases of the valvular apparatus		Valvular diseases.
Diseases affecting the pericardium		Chronic pericarditis. Hydropericardium. Hæmopericardium. Pneumo-hydropericardium. New formations on pericardium: cancer, tubercle, etc.
Congenital diseases		Abnormal positions. Closure of openings of right heart. Opening between the ventricles. Narrowing and closure of pulmonary artery, etc.

These are the organic diseases of the heart, save the rarest. But let us study the cardiac maladies according to their symptoms and signs rather than according to their anatomical classification.

Acute Diseases presenting Pain in the Cardiac Region; the Symptoms of a Disturbed Circulation; and a Change in the Sounds of the Heart, or their Replacement by Murmurs.

All the acute affections of the heart come under this head. In all, the sounds are either changed in their character or are replaced by murmurs. This is certainly true of endocarditis and pericarditis. All the acute disorders give rise, further, to more or less pain, and to anxiety of expression; in all there is fever; all are prone to occur in

connection with other morbid conditions, and especially with a contaminated state of the blood. In all, moreover, the symptoms of a disturbed circulation are met with: palpitation, irregular action of the heart, deranged flow of blood through the capillaries of different organs, and a tendency to dropsical accumulations. That these symptoms are not so clearly defined as in some of the chronic cardiac maladies is owing to the shorter time the complaint lasts.

Acute Endocarditis.—Acute inflammation of the lining membrane of the heart is very rarely a primary disease. It sometimes results from violent efforts, or from blows or other injuries to the chest. It is often connected with an acute infective process or a vitiated condition of the blood, as in pneumonia, in chorea, in cancer, in scarlet fever, in pyæmia, in puerperal fever, in Bright's disease, or in diabetes. But its most frequent association is with articular rheumatism.

The chief source of danger in endocarditis is the tendency the inflammation has to limit itself. It is confined to, or is most strikingly developed at, a part which bears least of all any impairment,—at the valves,—and often leaves behind it some permanent disorganization of their delicate structure. But it does not generally affect the entire valvular apparatus: that of the left side is usually alone the seat of disease. What morbid anatomy thus teaches, explains the occurrence and situation of the principal sign by which endocarditis is recognized. The roughness of the surface over which the blood flows, the minute vegetations, interfering with the function of the valves, occasions a distinct murmur, which is mostly confined to the mitral and aortic openings; it may be preceded by an altered character of the first sound or its reduplication.

Besides this blowing sound, there are other signs worthy of note. It is true, they do not form so leading a feature of the disease; still, they aid in its correct appreciation. The excited heart beats with augmented force, and sometimes with great irregularity, as the not unusual doubling of the second sound at the base proves. The size of the organ is not notably increased, except in those cases in which its cavities are choked with blood or fibrin-clots. The pulse corresponds to the action of the heart; yet not so closely as might be expected. It is, for the most part, frequent and strong. It becomes irregular, one beat being strong, the next weak, if the circulation through the heart be seriously obstructed; it may be feeble while the heart is thumping with violence against the walls of the chest.

The general symptoms are not uniform. There is usually a sense of uneasiness around the heart, with a fever showing a temperature

ranging from 101° to 103°, a short cough, palpitation and some irregularity of cardiac action, difficulty of breathing, and anxiety depicted on the countenance. To these are not uncommonly added turgescence of the face, headache, slight delirium, gastric irritability, diarrhœa, and rigors, followed by sensations of heat. Pain in the heart is rare, and is not likely to happen unless the pericardium or the muscular walls be implicated. In some cases an eruption of subcutaneous fibrous nodules occurs, especially in the rheumatic endocarditis of children.

Now, where these symptoms are present; where they manifest themselves in one whose system is in a state in which endocarditis is apt to take place; and where they are accompanied by a blowing sound recently and rather suddenly developed,—we are certain that inflammation is working its changes in the lining membrane of the heart. Yet some circumspection is requisite before arriving at this conclusion. A murmur may be attended with febrile signs and not be dependent upon acute endocarditis. The sound may be of organic origin and chronic; or it may be engendered in the course of an idiopathic fever, and the lining membrane of the heart be unaltered.

In the first instance the murmur is *old*, and results from some chronic injury to the valve, the attending fever being an accidental complication. Here is undoubtedly a difficult case for diagnosis. We see the patient for the first time; he has fever; his heart is acting strongly: a distinct blowing sound is perceived over it, How are we to tell that his complaint is not acute endocarditis? We have no absolute means of deciding that it is not. Yet by careful inquiry we can usually come to a knowledge of the truth. If the patient do not recollect to have suffered previously from dyspnœa or palpitation; if the cardiac excitement be well defined; if the face denote distress; if the accompanying symptoms indicate a state that is prone to be complicated with endocardial inflammation,—it is this disease under which he is laboring. Then the murmur is not so rough, is not often heard except during the systole, and may be changeable in its seat, which an old-standing murmur never is. Besides, it is not associated with those signs of enlargement which are invariably found when the valves have been for any length of time affected, unless the acute inflammation occur in a heart the valves of which have been previously spoiled. Under such circumstances, we can only conjecture what is going on within the organ from its increased excitement, and, if I may take my own experience as the general rule, from the character of the blowing sound undergoing alteration. It is rendered often less distinct, nay, it is even entirely muffled, by the products of the recent inflammation.

But how are we to distinguish between the soft murmur arising in the *course of fevers*, and that resulting from effused lymph? It, too, is not rough. It, too, happens with the impulse. It, too, is preceded by a lengthening of the first sound. Here is assuredly a strong resemblance; yet by no means an identity. The blowing sound in fevers does not exist until the blood is profoundly altered. In endocarditis it takes place almost as soon as the disease begins. The heart in fevers is not so directly disturbed in its action, and we do not find symptoms, local as well as general, which show that the circulation is obstructed. The blowing sound is rarely at the apex, but more over the body of the heart. To this some weight may be attached, since the murmur of endocarditis is very apt to be heard at the apex. But to no fact ought as much weight to be attached as to the one first mentioned, that the murmur takes place early and not late in the disease.

Throughout this description of endocarditis, only simple, uncomplicated cases have been kept in view; yet it is not often that the malady is seen in so pure a type. It is more generally accompanied by the friction sounds and other signs of acute pericarditis, and by the swollen joints, the painful movements, the acid perspirations, of acute rheumatism; or by the characteristic appearances on the skin of erythema marginatum; or by tonsillitis; or by the kidney symptoms of Bright's disease; or by the evidences of chorea, or of gonorrhœa, pyæmia, or septicæmia.

Nor is a murmur in endocarditis invariable. When the seat of the inflammation is not near the valves, a murmur is not generated. There may be also none if no vegetations exist on the valves, and perhaps in states of the exudation with which we are at present unacquainted. We cannot, under such circumstances, detect an attack of endocarditis. Yet it may be even then strongly suspected to be present if great excitement or irregularity of the heart manifest itself in a person who is laboring under a disease which predisposes to endocardial inflammation, such as rheumatism.

Clots of fibrin may form in the heart, and they or the vegetations which stud the valves be washed into the circulation. The *formation of clots in the cardiac cavities*, if at all extensive, announces itself by a sudden appearance or a sudden augmentation of the symptoms of obstructed circulation and of marked dyspnœa; the pulse is frequent and feeble, the action of the heart becomes exceedingly irregular, its sounds are indistinct, or a more or less distinct murmur is heard, and the extent of the præcordial percussion dulness is increased. Great anxiety, nausea and vomiting, delirium, turgid veins in the neck, and

fits of fainting, are also among the manifestations of the clogged blood in the heart. Yet these phenomena are not absolutely distinctive, for excessive dilatation without heart-clot will give much the same; and Walshe records that the effects of a rupture of a sigmoid valve or of a tendinous cord, during the acute endocardial disease, will give rise to symptoms exactly similar to the obstruction of the circulation resulting from polypoid concretions in the heart. When these thrombi form from other causes than endocarditis, as from heart palsy or morbid states of the blood unconnected with inflammation, the symptoms are not different.

Portions of the clots, or of the vegetations on the valves, are sometimes washed into the current, and the *embolism* occasions symptoms that, before we were aware of the damage to which the detached masses may give rise, appeared inexplicable. But now—when we see the circulation speedily diminished or arrested in a limb, and the limb becoming painful, swollen, or beginning to mortify; when we find that the flow of the blood through the brain has become suddenly disturbed, and the muscles of one side drop paralyzed; when the difficult breathing becomes rapidly still more difficult, while there are no signs of a superadded affection of the lung, nay, while the power fully to expand the lungs remains unimpaired, or while an effusion of fluid into the air-vesicles follows the dyspnœa —we know what has happened: we know that a broken-off piece of fibrin has been driven into the artery of the limb, or into the brain, or into the branches of the pulmonary artery, and, being too large to go any farther, has stuck fast, and has given rise to all these sudden and sad consequences. Sad indeed they are; for, even if the plugs do not lead to an immediately fatal result, they lay the groundwork for structural alterations in any tissue in which they become impacted.

Inflammation of the aorta may occasion many of the symptoms of acute endocarditis; at all events, it may do so when the upper part of the aorta is implicated. But it is not a condition that can be discriminated with certainty. The most significant signs are hurried respiration, a sharp, rapid pulse, tumultuous action of the heart, pain in the præcordial region, often greatly increased by movements, and also felt along the course of the spine, burning pain referred to the sternum, great anxiety. The history of the case points to gout, alcoholism, syphilis, or malaria. There may be paroxysms of pain such as occur in angina and a loud systolic blowing sound. When the abdominal aorta is affected, we notice strong local pulsation, and a marked murmur will be heard with greatest distinctness at or near

the seat of the inflammation. In some cases of aortitis, Bright[1] observed an extremely high degree of morbid sensibility over all parts of the body, which caused the patient to scream with pain when his wrists were merely touched. The disorder is most apt to happen in cachectic persons; and it has been repeatedly observed in those attacked with erysipelas, or after operations and injuries.[2]

Dissimilar causes may lead to different sites of endocardial inflammation. Thus, puerperal endocarditis is apt to localize itself in the right heart. It has pulmonary complications, and the progress of the disease is often slow; it may last several months.[3]

There is a form of endocarditis which may be here briefly mentioned,—*ulcerative endocarditis*. It is not common in this country, although I have seen a number of instances. It occurs mostly in connection with low forms of rheumatism or with blood-poisoning, and the symptoms of this, or of pyæmia, or a low septic fever, are apparently the prominent features of the case, or it may happen as subsequent to pneumonia.[4] The ulceration perforates the valves, and may extend into the muscular structure of the heart; pneumonia or pleurisy, embolic formations, and infarcts and metastatic abscesses in various parts of the body are among the common attendants,—pneumonia is especially frequent. The perilous affection shows an endocarditis developing amidst the symptoms of profound blood-poisoning and prostration, although these physical signs may be masked by a pericardial complication. Marked and recurring chills, like those of malarial fever, but coming on irregularly; a temperature of 105° to 107°; an extremely rapid pulse, becoming suddenly much slower, though very irregular; profuse sweats; vertigo; delirium followed by stupor; dry tongue; vomiting and diarrhœa; jaundice; tenderness over liver and spleen; and scanty, albuminous urine,—are among the prominent features of the malady. As regards the thoracic symptoms, there may be oppression, dyspnœa, and pain, yet these symptoms may be wholly wanting. In some instances a peculiar diffused rose rash, here and there mixed with papules and spots of ecchymosis, is noticed; in others there are capillary embolisms. By some, ulcerative endocarditis is looked upon as diphtheritic; indeed, when it has happened during puerperal fever, diphtheritic exudations have been found on the mucous membrane of the vagina and uterus. It is certain that the pyogenic cocci are constantly present, generally

[1] Guy's Hospital Reports, vol. i.

[2] Chevers, *ibid.*, vol. vi., and 2d Series, vol. i.; Osler, Gulstonian Lectures.

[3] Luzet et Ettlinger, Archives Générales de Médecine, Jan. 1891.

[4] Archives de Physiologie, Aug. 1886.

streptococci, staphylococcci, and pneumococci, and are found not only in the heart, but also in the infarcts in the spleen and liver. Death is the common ending,—either by gradual exhaustion, or suddenly by the tearing away of the injured valves.

The disease is extremely rare in children. It is more often mistaken for typhoid fever than for any other disease. But it is also mistaken for typhoid pneumonia, for cerebro-spinal fever, and for hemorrhagic smallpox. When ulcerative endocarditis happens in connection with malarial poisoning, a not infrequent association in Africa, its seat of predilection is in the aortic valves.[1] The most common type of the disease is the typhoid type. The malignant endocarditis may become engrafted on a chronic valve lesion. Its clinical association with a suppurative wound or puerperal disease is common, and we find it also in abscesses in the throat, and in combination with suppurative meningitis. The cardiac symptoms may be very obscure, and the occurrence of embolism during a febrile process be the first sign to explain their meaning. Rigors are common, and are the cause of malignant endocarditis being frequently mistaken for malarial fevers. High fever is the rule, and is an important element in the diagnosis. But I have met with instances, proved such by the autopsy, in which fever was almost absent.

Acute Pericarditis.—Acute inflammation of the serous membrane of the exterior of the heart is very similar to that of its interior. It is developed under the same circumstances. It is found in rheumatism, in gout, in Bright's disease, in scurvy, in alcoholism, in scarlet fever, in septic processes, or as an extension of inflammation from pleuro-pneumonia; it is very rarely idiopathic. The pericardial malady exhibits the same frequent association with rheumatism as the endocardial malady; it presents the same symptoms. Nature has not, indeed, drawn a very strict line of demarcation between the two diseases. When one exists, the other is very apt to attend it. Yet we do meet with endocarditis without pericarditis, and more often still with pericarditis without endocarditis.

The anatomical effects of inflammation of the pericardium are like those of acute pleurisy. The pericardium becomes injected and dry; plastic lymph accumulates on its surfaces, and especially on the surface which fits tightly around the heart. This stage of the disease corresponds to the dry stage, or plastic stage, of acute pleurisy. It may have the same termination by the two roughened surfaces adhering. But it is often followed by a stage of effusion. The effusion

[1] Lancereaux, Arch. Gén. de Méd., April, 1881.

may remain stationary or be absorbed, and the rugged portions of the membrane be placed again in apposition.

The characteristic sign of the plastic stage is a friction sound. Yet the friction sound is not always the same in extent or in character, because the deposited lymph is not always the same in extent or in character. The sound is like the crumpling of parchment, or the creaking of new leather, or it is grazing, or like a series of irregular clicks. It is single or double, and is prone to mask the natural sounds of the heart. But these are all points which have been already described: we shall merely add that when the friction develops itself

FIG. 45.

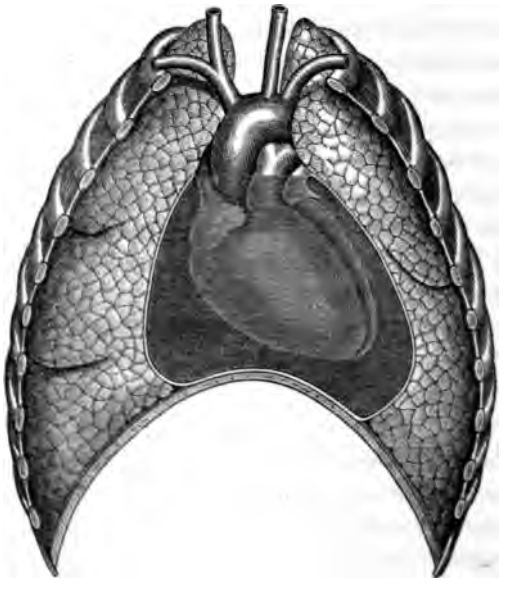

Illustration of the position of the heart in pericarditis, and of the distention of the pericardium with fluid. The heart-sounds are indistinct, except above the effusion; the impulse is feeble. The extent and shape of the percussion dulness may be judged of by the appearance of the distended sac.

under our observation, and with signs of excitement of the heart, it is as distinctive of inflammation of the pericardium as a recent blowing sound is, under the same circumstances, distinctive of inflammation of the endocardium. When the pericardial effusion takes place, it ceases; but only gradually, and not always completely; and in any case it is not uncommon for the ear still to recognize the friction sound at the base of the heart and around the origin of the great vessels.

The percussion dulness due to the effusion is generally considerable; and its contour is characteristic. When the patient is in the

erect posture, it is pyramidal; when he lies on his back, or changes from side to side, the outline of the flat sound is somewhat altered. Rotch,[1] in an elaborate inquiry into the matter, points to the dulness in the fifth intercostal space to the right of the sternum as occurring even in small effusions; and Roberts,[2] in his excellent monograph, speaks of the valuable aid afforded by it to surgeons about to tap the pericardium. Another significant sign connected with the dulness is that, as Bamberger has taught us, an area of dulness near the angle of the scapula which coexists with bronchial breathing and increased fremitus, and which is perceived when the patient is erect, is greatly influenced by position. It disappears, and with it the other signs mentioned, as he leans forward, to return as the erect posture is resumed.

In cases of considerable effusion, the intercostal spaces of the cardiac region widen, the eye recognizes a distinct bulging, and the dulness on percussion reaches far upward, to the second, or even to the first, rib. Within the space of dulness is sometimes seen an irregular, wavy motion; and what the eye detects the hand feels. But no movements, or only slight movements, may be perceptible in the præcordia. The heart, with its point pushed upward and outward by the accumulating liquid, has to struggle to reach the walls of the chest. Its contractions are irregular; its impulse is feeble, or all appreciable impulse has ceased. The sounds heard through the mass of fluid seem distant and muffled. Yet the second sound over the upper part of the sternum and at the base of the heart retains its sharpness.

During the stage of absorption the apex returns to its natural position; the dulness gradually disappears; the sounds and the impulse regain more of their normal character; the friction murmur reappears, and then ceases, leaving not infrequently the two surfaces of the pericardium adhering.

We cannot foretell how long it will take the disease to run through its different stages. Death may occur in less than thirty hours, the heart being paralyzed by an enormous effusion; on the other hand, the acute attack may last for as many days, and then leave serious traces. But whatever stage the malady be in, it can be recognized only by the physical signs: by the friction, the peculiar percussion dulness, the enfeebled impulse and heart-sounds.

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[1] Boston Med. and Surg. Journ., 1878, vol. xcix.; also article "Diseases of the Pericardium," in Keating's Cyclopædia of the Diseases of Children, vol. ii.

[2] Paracentesis of the Pericardium, Phila., 1880.

There are no general symptoms that prove a pericarditis to exist. There are symptoms by which we may infer that pericarditis is present; but there are none which absolutely belong to it and would prevent it from being overlooked. The symptoms usually met with are those of inflammation of the endocardium, but with more decided local evidence of disorder. We find the anxious expression; the fever, not generally high; the œdema; the same uncertain or irregular pulse. But there is more pain over the heart,—acute, severe pain, shooting to the left shoulder, augmented by movement, increased by pressure, and associated with epigastric tenderness; there is more dyspnœa, because the distended sac presses on the lung; a dry, irritative cough; and sometimes difficulty in swallowing. Yet every one of these symptoms may be absent. The pulse may be regular; the breathing not perceptibly accelerated or laborious; and even the important symptom of pain, though this is rare, may be wanting from the beginning to the end of the disease.

When the action of the heart grows weaker and weaker, the circulation becomes more irregular. The beat of the artery at the wrist is feeble, and intermits; the veins of the neck are prominent; the skin is cold and pale; the extremities are œdematous. These are always symptoms of grave import.

If next we inquire with what complaints acute pericarditis is likely to be confounded, inflammation of the endocardium and inflammation of the pleura occur at once to the mind. To contrast the signs of the first two maladies, for the slight difference in their symptoms has already been mentioned:

Endocarditis.	Pericarditis.
Blowing sound; excited action of the heart.	Friction sound; excited action of the heart.
Slight, if any, increase of percussion dulness.	In stage of effusion, marked and extended percussion dulness.
Impulse strong.	Impulse wavy and feeble.
Sounds normal or more distinct, except at site where murmur is heard.	Sounds feeble and muffled, except at base; no blowing sound.

Such is the distinction of pure cases of each disease. Still, as already stated, the affections are often combined. It is not uncommon to hear with the friction sound a distinct endocardial murmur. But there is sometimes a difficulty of another kind in the way of a precise diagnosis. The murmur produced on the outside of the heart may simulate so closely the murmur produced in its interior that it is almost impossible to discriminate between them. The

former may completely possess the blowing characters of the latter. Mostly, however, it is rougher; more prone to be double; and each division is like the other, equally rough, equally superficial-sounding, equally lacking in strict correspondence to the systole or to the diastole. And, above all, the sound alters at times both in situation and in character with amazing rapidity. Perceived now as an ordinary bellows murmur on the left side, it is after the lapse of some hours heard as a rough rasping sound on the right. These changes have a high degree of value. But they are not of constant occurrence; and to say that it is sometimes impossible to tell a pericardial from an endocardial sound is to say no more than is borne out by every-day experience. In the stage of effusion pericarditis is not likely to be mistaken for endocarditis.

Pleurisy gives rise to some of the same symptoms and signs as pericarditis. It develops a friction sound; it occasions dulness on percussion, dyspnœa, and cough. But the physical signs are in different situations. In the one disorder they are in the region of the heart, and are confined there; in the other, they are spread over the whole side of the chest, and are most perceptible at the back. This is true of the dulness, and, for the most part, of the friction sound, which, when of pericardial origin, is rarely heard posteriorly.

At times, however, we meet with very puzzling cases. A friction sound discerned over the heart may be in reality produced *in the adjoining pleura*. The patient is directed to suspend his breathing; the friction sound does not stop. Now, the inference from this would be that the sound originates in the pericardium; and in the large majority of instances this is a correct inference. But it is not always so. The friction may have its seat in the pleura and be caused by the movements of the heart. There are no absolute means, besides the intermission of the sound during some of the beats of the heart, as well as during some of the acts of breathing, especially in expiration, of detecting in these rare cases the true seat of the disease. Then, both in pleuro-pneumonia and in phthisis there may be a *pleuro-pericardial friction*, from an attending pericarditis. It also is much influenced by the respiratory acts. During deep inspiration it lessens or disappears; expiration intensifies it.

To confound the dulness on percussion caused by liquid in the pericardium with that due to *liquid in the pleura*, is a mistake the more likely to happen, because the two serous membranes, and indeed the lung, are often involved in the same inflammation. But a pericarditis uncomplicated with pleurisy or with pleuro-pneumonia does

not change the clear sound at the back of the chest save in rare cases of enormous accumulation of fluid. Effusion into the pleura gives rise to a flat sound anteriorly; to a still more perceptible dulness at the inferior portion of the chest posteriorly; and the sounds of the heart remain unaltered.

These, then, are the diseases with which acute pericarditis is liable to be confounded. There are several chronic cardiac maladies which will occasion some of the same signs and symptoms: such are thinning of the ventricles with distention of the cavities, and a dropsy of the pericardium. But the history of these affections is different, and their signs, although similar, are not precisely the same. The *dropsy of the pericardium* is associated with dropsies elsewhere, and with some obvious cause accounting for the watery effusion, and at no stage of its existence does it exhibit a friction sound, while albumin in the urine, œdema of the lungs, or hydrothorax are common attendants. A double friction sound at the right base may cause a plastic pericarditis to be mistaken for *aortic regurgitation*. But the marked coexisting hypertrophy in this affection, the unchanging character of the abnormal sounds, and the peculiar pulse, guard against error.

There is another complaint of which pericarditis sometimes borrows the garb. The thoracic symptoms may be latent, but the disease may produce the symptoms of extreme *gastric irritation* or inflammation. Nausea and vomiting are marked, and tenderness on pressure in the epigastric region. All the remedies are directed to the stomach; and at the post-mortem examination the physician stands amazed at finding this viscus healthy and the pericardium full of serum or pus. An inquiry into the state of the heart might have saved him from a serious blunder.

Another grave error which may be thus obviated is the mistaking of some cases of acute pericarditis, on account of the wild delirium they present, for acute *inflammation of the brain*. Now, both in endocarditis and in pericarditis this active delirium may throw all the other symptoms into the background. It is difficult to see why a pericardial inflammation should give rise to such violent disturbance of the brain. It is not at all unlikely that it has its origin, in part, at least, in the contaminated state of the blood which occurs in the affections, as rheumatism or Bright's disease, with which pericarditis is often associated. However occasioned, it is necessary to be aware that the cerebral symptoms arising in inflammation of the membranes of the heart may entirely draw off attention from the serious lesions within the chest. A fixed delusion of having committed some crime appears

to Flint[1] to be a distinguishing feature of the mental wandering; while Sibson[2] in his exhaustive analysis points out, what I have known to happen in more than one instance, that the desponding and taciturn, or, as he calls it, sombre delirium lasts from two or three weeks to as many months.

Can we by the symptoms or physical signs tell the character of the fluid in the sac? We cannot by the signs; and by the symptoms we can only suspect pus if there be recurring chills, and irregular but high temperature, and if the pericarditis have arisen in the course of a malady that makes the presence of pus likely. *Hemorrhagic pericarditis* can also only be distinguished as a probability by the history. It happens in scurvy and in purpura, and may be an attendant upon tubercle or cancer of the pericardium. *Cancerous pericarditis* produces also serous or purulent effusion. It is never a primary disease, and it has no characteristic symptom, except it be, in some cases, darting pain in the præcordial region attending the signs of pericarditis. It is by the history and the evidence of deposit elsewhere that we have to judge. The same is true of *tubercular pericarditis*. Here the pericarditis is often dry, and the membrane much thickened. Yet an enormous effusion may occur, as happened in a case recorded by Musser.[3]

Let us now inquire in how far one of the terminations of pericarditis by adhesion or agglutination of the surfaces can be recognized. In many of such cases, whether there be coexisting dilatation, or hypertrophy, or what is most common, combined dilatation and hypertrophy, we find changed rhythm and dyspnœa, œdema of the extremities, and syncopal attacks. Yet these are not special signs of pericardial adhesion. Indeed, there is not a single symptom or sign constant, or by itself characteristic of pericardial adhesion. The most trustworthy signs are a drawing in of the apex of the heart during the contraction of the ventricles, with a depression in the intercostal spaces becoming visible at the same time, and sometimes with a simultaneous sinking in at the lower half of the sternum; the limits of the increased dull percussion sound in the præcordial region remaining unaffected during inspiration and expiration; a fixed apex beat, uninfluenced by change of posture of the body or by the acts of breathing; diminution of the inspiratory movements in and near the epigastrium; greatly extended undulatory impulse; and diastolic

[1] Diseases of the Heart.

[2] Article "Pericarditis" in Reynold's System of Medicine.

[3] Medical Diagnosis.

rebound felt on placing the hand over the seat of the impulse. Enfeeblement or absence of impulse, while it may happen, is much rarer. A sign of value is the one pointed out by Broadbent, a drawing in with the systole of the posterior and lateral walls of the chest, generally most evident between the eleventh and twelfth ribs, and indicative of universally adherent pericardium. Duroziez[1] attaches importance to the nipple being kept in constant motion. Friedreich[2] dwells on a rapid emptying of the veins of the neck during the diastole of the heart, while with the systole they swell up; and Riess[3] tells us that, owing to the close bringing together of the heart, diaphragm, and stomach, the heart-sounds resound with a metallic ring. The heart-sounds, owing to the frequent association of adherent pericardium with valve affections, may be replaced by murmurs. To the occurrence of a presystolic murmur, Hale White has called special attention. When the pericardial surfaces are extensively and firmly united, the eye is struck by the evident depression of the præcordial region. When the pericardium is adherent to the sternum and bands pass off compressing the aorta, "indurated mediastino-pericarditis," a pulse vanishing with each full inspiration—*pulsus paradoxus*—has been described by Kussmaul.[4] The same sign has been noticed by Irvine in cases of adherent pericardium and pleura, and by Traube[5] in exudative pericarditis where the mediastinum was not implicated. Aran has proved the tendency to sudden death in complete pericardial adhesion.

Closely connected with the subject of inflammation of the pericardium is that rare affection in which air is present in the pericardial cavity, *pneumo-pericardium*, or, more strictly speaking, on account of the frequent association with fluid, *pneumo-hydropericardium*. It occurs as the result of injuries, of communication established by disease between the pericardium and the neighboring organs, and in very exceptional instances is due to decomposition of liquids in the sac. Its chief diagnostic features are abnormal resonance over the cardiac region, and a metallic character of the heart-sounds. The tympanitic resonance alters in a most marked manner with changes in the posture of the patient, and is limited by a distinct line of dulness caused by the fluid. The metallic sounds may at times be heard at a distance, and may be attended with sounds of most extraordinary kind, friction

[1] Traité clinique des Maladies du Cœur.

[2] Virchow's Archiv, Bd. xxix.

[3] Berliner klinische Wochenschrift, No. 51, 1878.

[4] Ibid., No. 37, 1873.

[5] Charité Annalen, 1876.

sounds mixed with splashing and gurgling, the so-called water-wheel sound, the *bruit de moulin;* generally an intermittent sound, at first metallic. The cardiac impulse is feeble or absent. The symptoms of pneumo-pericardium are vague, generally those of a pericarditis, with great difficulty in breathing, high, fluctuating temperature, chest pain, and failing circulation. In point of diagnosis we must be careful not to be misled by the modification of the cardiac sounds and the splashing and metallic phenomena due to a dilated stomach. From *pneumothorax*, even when encapsulated near the heart, we distinguish pneumo-pericardium by the dulness on percussion to be found over the displaced heart in the former malady, and the amphoric or metallic respiratory sounds that are heard in addition to the metallic heart-sounds.

The discovery by Welch of the bacillus aërogenes capsulatus, and its association with gas forming in the tissues and cavities, will explain instances of pneumo-pericardium following wounds. The entrance of air may happen, as in the cases of Meigs[1] and of Müller,[2] by a rupture brought about by the pericardial exudation,—in the one case into the œsophagus, in the other into the lung. These cases of ulcerative perforation almost all end fatally.

Myocarditis.—Of inflammations of the substance of the heart there are two chief varieties,—the acute inflammation of the muscular walls, and the chronic myocarditis or fibroid degeneration. The *acute* gives rise to infiltration between the fibres of the heart of blood-corpuscles, of proliferating cells, and of leucocytes, and the muscular fibres themselves become granular and degenerate. Local softening and circumscribed abscess, and even gangrene and perforation of the ventricle may result. But we are not enabled to foretell the state of the heart during life, mainly because the muscular structure is rarely affected without the endocardium, or still more frequently the pericardium, being implicated, and thus the manifestations of these disorders occur mixed with those of the myocarditis. Great pain in the cardiac region is the most usual and the most prominent of the symptoms. The breathing is generally much oppressed; delirium is often present; the urine is scanty and albuminous; the heart fails in power; and the patient dies in a state of utter prostration or suffocates from pulmonary œdema. The pulse, as in endocarditis or in pericarditis, exhibits no uniform character. The statement that it is invariably intermittent, feeble, and quick, is not correct. It is so as the disease

[1] Amer. Journ. Med. Sci., Jan. 1875.

[2] Deutsches Archiv für klinische Medicin, Bd. xxiv., 1879.

advances, but it may be full, and not above eighty, long after the distress in the chest is unbearable.[1] The temperature may be only slightly elevated or very high. The signs of cardiac failure are quickly developed. The heart-sounds are weak and irregular, and, owing to acute dilatation occurring, the cardiac dulness increases. In purulent myocarditis the temperature shows marked remissions and exacerbations, and rigors and sweatings are usual.[2] Acute myocarditis may occur in rheumatism, but it is most common in pyæmia and in phlebitis. Its occasional association with gonorrhœa has been pointed out, and it may be found with or without gonorrhœal rheumatism.[3] In children there is a distinctly cerebral form.[4]

Acute interstitial myocarditis and parenchymatous myocarditis, the muscular fibres in both being infiltrated with granules, have no distinctive symptoms. They occur in fevers, particularly in typhoid fever, yellow fever, and smallpox, and in pericarditis, and may be suspected under these circumstances from the feeble heart action.

Chronic myocarditis, or fibroid degeneration, often results from rheumatism, or attends pseudo-hypertrophic paralysis. A very common cause is disease of the coronary arteries, especially obliterating endarteritis of syphilitic origin. The disease is most common in men, and may lead to aneurism of the heart. The diagnosis of chronic myocarditis is as uncertain as that of the acute form. The symptoms are those of a feeble heart: œdema, breathlessness on exertion, cough, hemorrhages into different organs, venous congestions, hydrothorax, occur. In some cases there is pain over the heart or marked anginous attacks occur. The percussion dulness in the cardiac region is somewhat increased, and the heart is generally dilated, or in a state of combined dilatation and hypertrophy. The first sound is indistinct, or there is a mitral systolic murmur; the second over the aorta is apt to be accentuated or doubled. A significant sign is a want of correspondence between the heart and the pulse-beats; these are unequal and irregular.[5] Some stress may be laid on signs of pericardial adhesion, if present.

[1] Salter, Medico-Chirurgical Transactions, vol. xxii. In several of the cases on record, for instance in the one mentioned by Graves in his Clinical Lectures, there was coexisting valvular disease, which, of course, invalidates the statements as regards the character of the pulse, and, indeed, as regards many of the other symptoms.

[2] Bramwell, Diseases of the Heart, Edinb., 1884.

[3] Councilman, Amer. Journ. Med. Sci., Sept. 1893.

[4] Mitchell Bruce, Keating's Cyclopædia of the Diseases of Children, vol. ii.

[5] Ruhle, Archiv für klin. Med., 1878.

Chronic Diseases attended with Increased Extent of Percussion Dulness, but with Normal or almost Normal Heart-Sounds.

To this group belong those diseases which affect the walls of the heart or its cavities, without having involved the valvular apparatus, such as hypertrophy and dilatation,—types of the two different states of force and of weakness, but both exhibiting an extent of percussion dulness greater than in health, and heart-sounds not materially changed.

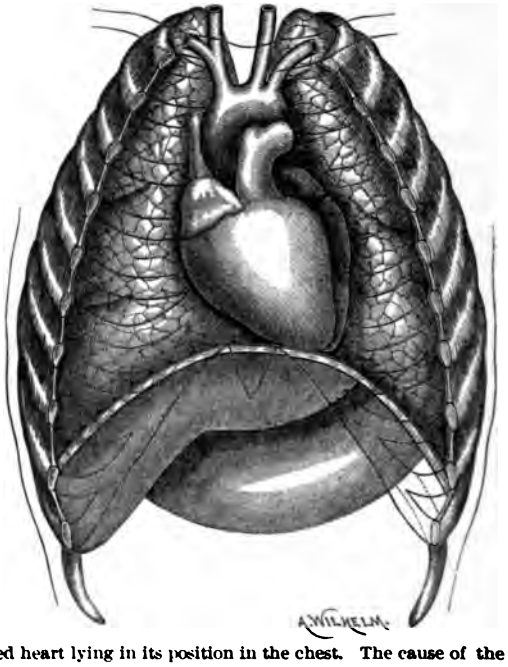
Hypertrophy.—Hypertrophy of the heart is an overgrowth of its walls, and usually also of its cavities; for, although we may have the muscle thickening without the cavity enlarging, nay, even with it diminishing in size, neither this simple nor the concentric hypertrophy occurs, save in rare instances. It is evident that any one of the chambers of the heart may alone become hypertrophied. But, practically, the state we mean when speaking of cardiac hypertrophy is an increase of the ventricles, and especially of the left ventricle, in its wall and cavity, with a similar, although much slighter, expansion of the right side.

The physical and vital manifestations of the heart having outgrown its natural dimensions are these: The pulse is full and strong, and somewhat tense. The face is florid, or else it is pale; and the mucous membranes of the lips and eyelids are injected. The eyes are bright, and apt to be prominent. The carotids pulsate forcibly under the least excitement. Some persons suffer from headache and giddiness; in fact, all the symptoms denote a circulation actively—too actively—carried on. Yet the symptoms directly referable to the heart are not marked. There is, as a rule, no pain or irregular action of the heart, nor do violent fits of palpitation occur. What the patient comes to consult his physician about are rushes of blood to the head; or a ringing in the ears; or a feeling of weight in the epigastrium which troubles him after a full meal; or shortness of breath; or in consequence of the powerful action of the heart, when lying in bed, attracting his attention; or because he is alarmed about a dry cough, and believes himself the victim of pulmonary consumption.

The physical signs are more uniform than the symptoms. We observe a fulness or arching of the præcordial region, and an impulse, strong, heaving, and extended over several intercostal spaces. The apex does not strike the chest walls between the fifth and sixth ribs, but its beat is perceived lower down, usually an inch or more to the outside of the nipple line. The extent of percussion dulness increases,

both longitudinally and transversely; and particularly in the latter direction, if the right ventricle be much enlarged. This peculiarity in the expansion of the area of dulness on percussion forms, in truth,—with the greater dyspnœa, and with an impulse more directly perceived over the right side of the heart, near the pit of the stomach, and often out of proportion to the compressible and rather small radial beat, and with the increased distinctness of the second sound of the pulmonary artery,—the sign that hypertrophy with dilatation has principally affected the right side.

FIG. 46.



An hypertrophied heart lying in its position in the chest. The cause of the lowered apex beat, and of the extension of the impulse, as well as of the somewhat squarer outline of the increased dulness over the enlarged organ, is obvious from the shape and position of the heart.

The first sound of an hypertrophied heart is duller than in health, but prolonged and weighty. The second sound is not particularly changed. There are no murmurs, except under rare circumstances, which will be mentioned in discussing valvular diseases. Thus, the greatest value of auscultation is that, by showing the sounds but little altered, it enables us positively to exclude a lesion of the valves; just as the chief service of percussion, with reference to an enlarged heart, consists in permitting us to distinguish the excited motions of the simply disturbed organ from the action of a heart the walls of which are

thickened; and as the main use in noting the impulse is that it serves as a means of discrimination between hypertrophy and those affections in which the beat is weakened, such as dilatation or a pericardial effusion, or between the dulness in the præcordial region due to hypertrophy and that caused by deposits in the pleura, in the mediastinum, or in the lung. Where there is contraction of the left lung, as from pleurisy or fibroid change, more of the heart is exposed, and the dulness on percussion in the cardiac region is increased, as well as the impulse, which is felt over a larger space and to the left; but the cardiac sounds are unchanged, and deep inspiration alters the extent of cardiac dulness but little.

Hypertrophy may be combined with decided dilatation of the heart. This kind of hypertrophy presents a less dull, prolonged first sound, and the pulse, though full, is likely to be more compressible. Hypertrophy may affect specially any part of the constituents of the muscular walls. Thus, the connective tissue, as Quain has particularly called attention to, may be alone concerned in the morbid action. Hypertrophy of the heart is found much more frequently among males than among females. Its causes are various. It is common in Bright's disease and in general arterial sclerosis; continued functional excitement produces it; so does any kind of strain and overaction, and perhaps excessive nourishment. It is found to be common among inordinate beer-drinkers. But the main cause is an obstruction to the circulation, either in the heart or in other organs. It is for this reason that the complaint is so often met with in connection with diseases of the valves or of the large arteries, and that the right side of the heart enlarges when the pulmonary air-vesicles are over-distended. We also encounter hypertrophy in the heart as a consequence of obliteration of the pericardial sac. In the hypertrophy of chronic nephritis reduplication of the first sound is often noticed.

There is a form of hypertrophy of the heart to which attention has been particularly called by Fothergill's description,—the so-called *gouty heart.* Generally there is coexisting chronic contracting kidney. In the first stage we find decided hypertrophy with accentuation or booming of the second aortic sound, high blood-pressure, tense pulse, hardened arteries, and the passage of large amounts of pale urine of low specific gravity. The renal changes may or may not be evident; we may or may not detect albumin in the urine. In a subsequent stage of the malady there is failure of the circulation. The cardio-vascular phenomena are early made perceptible by the sphygmograph. The full, tense pulse gives a full up-stroke, a broad summit, and a

retarded down-stroke; the "square-headed tracing" formed is very characteristic of the malady, and bespeaks the fibroid change in the kidney, whether or not albumin be found. In some instances considerable cardiac dilatation as well as hypertrophy is present. The high blood-pressure is due to the waste-laden blood. The skin often exhibits little twigs of dilated vessels; the ear is usually deep red, with a large glistening lobe; or in spare persons the lobe looks withered; the teeth become blunt and worn down in time; the hair is apt to be iron-gray. There is the history of gout, acquired or hereditary, but there may have been no active outbreak of gout, rather the condition of imperfect assimilation known as lithæmia.

Dilatation.—Except in its seat in the ventricles, dilatation of the heart is the reverse of hypertrophy. The cavities are stretched out of all proportion to the thickness of the muscular walls; these may be slightly thicker than normal, or of natural thickness, or thinner, and apparently hardly capable of supporting the weight of the blood.

Almost opposite symptoms and physical signs to those of hypertrophy result from dilatation. In place of activity and power, everything indicates inaction and stagnation. There is a very strong tendency to venous congestions and to dropsies. The portal system is gorged. The liver increases in size. The bowels are constipated. The urinary secretion is interfered with, and sometimes albumin is passed. The hearing may become dull. The patient is languid and feeble, and his intellect obtuse. He suffers from chilly sensations, and from uneasiness in the cardiac region and palpitations. The pulse is small, unequal, and irregular, and the veins of the surface are swollen. The skin around the ankles, and often at other parts of the body, pits on pressure. But, since it is the right side of the heart which is usually the most affected, the lungs show most plainly the effects of the venous stagnation. Breathlessness on exertion or difficulty in breathing, making itself at times manifest in paroxysms attended with wheezing respiration; a chronic cough; a collection of serum in the pulmonary structure,—all add to the misery which the perilous malady entails. And as it is commonly some obstructive disease in the lungs, such as emphysema, which has given rise to the dilatation of the right side of the heart, so this again augments the morbid state of the lungs, and aggravates the symptoms.

The physical signs are very unlike those of hypertrophy. The same extended dulness on percussion exists; but it is associated with a feeble and fluttering impulse, which is in strong contrast with the heaving, powerful blow of an hypertrophied left ventricle, and which at times cannot be localized, or may be seen, yet cannot be felt. The

sounds in cardiac dilatation are not always the same. When the walls are thin, they are clearer, sharper, and more ringing than in health; if, however, the muscular structure be at all degenerated, the first sound is faint and very ill defined. The second is often split, giving rise to the so-called gallop rhythm. But no murmurs are perceived, unless a watery state of the blood produces them, or unless it happens—and it does not infrequently happen—that the dilatation of the heart is conjoined to valves incompetent, either temporarily or permanently, to prevent regurgitation.

FIG. 47.

A dilated heart, the right ventricle opened. In this case there was no valvular disease. Hence the characteristic physical signs; the increased dulness on percussion, the extended but weak impulse. The first sound was feeble, for the organ was flabby as well as dilated.

In *acute dilatation* of the heart, such as we sometimes see in fevers, or in pneumonia, or after violent exertion and strain, or from shock or sudden fright, or where an hypertrophied heart suddenly fails in power, we have, besides the symptoms of great venous congestion, dyspnœa, and rapid, feeble impulse, or impulse irregular, now strong, now weak, temporary systolic murmurs of varying site, chiefly a systolic apex murmur. But the murmur may be near the ensiform cartilage over the tricuspid area, or, as in a case observed by Broadbent,[1] over the pulmonary artery.

[1] Heart Disease, p. 241.

Dilatation is not always pure; it is met with in every possible degree, and in combination with hypertrophy and valvular diseases. Accordingly, its symptoms and signs are somewhat dissimilar. But one constant feature it preserves; it always holds up to view both the vital and the physical manifestations of a weak heart. Indeed, when an hypertrophied heart dilates, the signs of relative weakness become superadded, the impulse is not so strong as before in comparison with the percussion dulness, and dropsy becomes a marked symptom. Pure dilatation is likely to be confounded with the diseases in which enfeebled action of the heart is encountered, and these are fatty degeneration and a pericardial effusion.

Fatty Degeneration.—This is one of those disorders with the anatomical characters of which we are far better acquainted than with their clinical history. There is, indeed, no sign by which we can positively say that the dangerous disorganization of the muscular fibres of the heart is in progress. We may, however, suspect it, if the signs of weak action of the heart—feeble impulse and ill-defined sounds, especially the first sound—coexist with oppression, with a tendency to coldness of the extremities, with a pulse permanently slow and of low tension, or permanently frequent, empty and irregular, or rigid though weak, and be met with in a person who is the subject of a wasting disease, or has arrived at a time of life at which all the organs are prone to undergo decay. Something more than a probable opinion is warranted if, in addition, there be proof of sclerotic change in the vessels, or of fatty degeneration elsewhere, such as an *arcus senilis;* or if it be ascertained that the patient suffers from pain across the upper part of the sternum and from paroxysms of severe pain in the heart; that he sighs or yawns frequently; that he is easily put out of breath; that his skin has a yellow, oily look; that he is subject to syncope, or to seizures during which his respiration seems to come to a stand-still; and that he is liable to vertigo, to attacks of transitory unconsciousness, or to be stricken down with repeated attacks having the character of apoplexy, save that they are not followed by paralysis.

Now, here is certainly a group of phenomena dissimilar to those of a dilated heart. Let us add that the extent of the cardiac percussion dulness remains unaltered, except in those instances in which hypertrophy or dilatation coexists, that dropsies and local congestions are not prominent symptoms, or indeed do not happen at all, and the dissimilarity becomes still greater. A differential diagnosis would, under such circumstances, be anything but difficult. But in point of fact the matter is generally not so easily decided, and there are several

reasons why it is not. One is, that all the features described are rarely combined in the same case; indeed, one of the most marked, the Cheyne-Stokes breathing, is uncommon rather than common, and occasionally occurs in other cardiac maladies. The second is, because non-fatty softening, the result of a granular infiltration, as met with, for instance, in fevers, may present much the same vital and physical manifestations. The third is, because a fatty heart has a tendency to become dilated, and the symptoms and signs of the former disease are then merged into the symptoms and signs of the latter. With the organ in such a condition, the practical value of a differential diagnosis is, however, not great. Decided dropsy would indicate that dilatation had happened.

The remarks about fatty heart apply particularly to that variety in which the muscular structure in middle-aged or elderly persons has slowly undergone decay, and which is especially seen in men of sedentary habits, in tipplers, in the gouty, or in diabetics; disease of the coronary arteries often coexists. But we meet with fatty heart, although far less frequently, in young persons, and in a more acute form; and we encounter it in chlorosis, in pernicious anæmia, after repeated hemorrhages, and after phosphorus poisoning. Poisonous doses of acids, such as nitric, sulphuric, oxalic, are said by von Dusch also to give rise to the cardiac change.

Persons who have fatty hearts are subject to attacks of faintness, preceded or attended with *sensations of great coldness*, or a chill. Sometimes these attacks happen daily, or every few days, and in such a manner as to give rise to the impression that they are due to malaria. A number of instances of the kind have come under my observation, and I have met with them particularly at the end of fevers or other debilitating diseases happening in those affected with feeble hearts. The seizures, though bearing a resemblance to intermittent fever, are unlike it in being associated with signs of great weakness of the circulation or heart failure, sometimes joined to a sense of impending dissolution; in their irregular accession; and in their not being followed by fever. In doubtful cases the thermometer by showing the absence of the great rise of temperature of the malarial disorder, will materially assist us in the diagnosis.

Heart starvation, to which Fothergill[1] has called attention, has, in the feeble circulation, the cold extremities, the tendency to vertigo, and the pseudo-apoplectic attacks, symptoms common with those of fatty heart. But the malady is not associated with disease of the

[1] Edinburgh Medical Journal, May, 1881.

arteries, and is often an attendant upon general ill nutrition, and worry, and long hours of work and short hours of sleep.

A fatty heart sometimes *ruptures*. The symptoms that are mostly noticed are these: the patient is suddenly attacked with intolerable anguish in the heart; he presses his hand to it, then faints, and soon expires. Or else he lives for a short time, suffering from faintness, cramps, and difficulty of breathing, and with death plainly written on his face.

Chronic myocarditis with fibroid changes in the heart walls cannot be distinguished with any certainty from fatty heart. Extensive arterial degeneration, accentuation of the second aortic sound, signs of hypertrophy, attacks of palpitation and constant pain in the region of the heart would be in favor of cardiac fibrosis. But not one of these symptoms is convincing proof.

Where there is *fatty accumulation on the heart*, without fatty change of its fibres,—a condition we sometimes find in fat persons whose internal viscera are loaded with fat,—the manifestations are those of a feeble heart, and different from fatty degeneration only in degree. The first sound of the heart is weak and toneless: the pulse is feeble, but, as Walshe tells us, regular. The percussion dulness in the cardiac region is somewhat increased. A sensation of oppression over the region of the heart, or even actual pain, is complained of. There is shortness of breath on taking exercise and sometimes pretibial œdema. Fatty infiltration may be followed by fatty degeneration.

Of *atrophy of the heart* we know very little. All we know is that at times in certain wasting diseases, such as tubercular phthisis, cancer, and suppurating bone affections, the heart atrophies; it may also do so when the pericardium is tightly adherent; and cardiac atrophy is said to happen occasionally after pregnancy and chlorosis. It has not a single symptom nor a single sign by which it can be recognized with certainty. Diminished percussion dulness, clear sounds, and feeble impulse might enlighten us; but, even in cases where we have not been misled by emphysema of the lungs, or there is no coexisting fatty change, they are too uncertain to be made a basis for diagnosis, or attending lung conditions throw doubt on several of them. There is great tendency to palpitation, and the pulse, Hayden tells us, is quick, all but inappreciable, yet regular. The X-rays would furnish a valuable means of diagnosis.

Pericardial Effusion.—Pericardial effusion also presents the signs of a weak heart with increased dulness on percussion in the cardiac region, and is liable to be mistaken for dilatation of the organ.

But though there are points of resemblance to a dilated heart, there are points of contrast, as the subjoined table shows:

Dilatation of the Heart.	Chronic Pericarditis with Effusion.
Percussion dulness increased in extent, but square in outline.	Percussion dulness increased, but often of pyramidal shape.
Impulse in epigastrium.	Impulse in third or fourth left interspace, apex tilted upward.
Heart-sounds clear and sharp; sometimes, however, feeble.	Heart-sounds feeble and distant-sounding at the apex, but distinct near the upper part of the sternum.
No friction sound.	Often friction sound still heard at base.
Dropsy; signs of venous stagnation; severe cough, and dyspnœa.	Neither dropsy nor venous stagnation. Cough and dyspnœa are not such prominent symptoms.
The history of the disease shows it to be gradually developed.	The history frequently points to the acute attack.

Diseases of the Heart exhibiting more or less of the Signs and Symptoms of Enlargement of the Organ, and accompanied by Endocardial Murmurs.

Valvular Affections.—These, when not due to congenital malformations, are most commonly the result of rheumatic endocarditis, of slowly progressing sclerotic changes, or of heart-strain. A certain number of cases have their origin in some of the fevers, as in scarlet fever, and in septic conditions and blood-changes, as in malignant endocarditis. The different valves are not affected by these causes alike. Rheumatic endocarditis is the principal cause of disease of the mitral valve, especially of mitral insufficiency; but among prominent causes of this are also alterations in the muscular wall of the ventricle or in the tendinous cords. Aortic insufficiency is generally due to slow sclerotic changes in the valvulets, whether attended with atheroma or not, or to subacute or chronic endocarditis from heart-strain; it may be also owing to the sudden rupture of a valve previously damaged. Mitral constriction is mostly brought about by atheromatous or calcareous alteration, as is aortic constriction; but in mitral constriction we may have also a history of endocarditis in early childhood subsequent to rheumatism, an exanthematous fever, or chorea. In insufficiency of the tricuspid valve we can trace usually the result of over-distention of the right heart, such as follows pulmonary congestion caused by mitral disease, or of an obstructive disease of the lung, such as emphysema or cirrhosis. Tricuspid stenosis, and the other very rare valvular affections of the heart,—those of the pulmonary artery,—are commonly congenital.

To find the sounds of the heart clearly and well defined, is to know that no disease of the valve exists. When the valvular apparatus is disordered, the mischief betrays itself, for the most part, by a murmur. If, therefore, a murmur of any permanence be met with in the heart, especially if it be associated with the signs of either hypertrophy or dilatation, the inference that valvular disease exists will in the vast majority of cases be correct.

Yet it will not be so always; for there are other morbid states besides valvular affections which engender a murmur, which may be even accompanied by all the manifestations of enlargement of the heart. Malformations, such as communications between the auricles or between the ventricles, or between the great vessels near their origin, or impoverished blood, or a misdirected blood-current, may occasion a murmur.

Now, with reference to *malformations*, their presence in adults, or in children that have passed the days of infancy, is exceedingly rare. The most trustworthy symptom they present is that indicating the admixture of arterial and of venous blood; in other words, the symptom of cyanosis, the bluish discoloration of the skin. In addition, we may perceive clubbing of the nails, a tendency to hemorrhage, breathlessness, or dyspnœa, cough, and irregular action of the heart, and a blowing sound in the cardiac region; hypertrophy of the heart, especially of the right heart, is also very generally present. Still, the recognition of these malformations is always more or less a matter of conjecture. With the aid of more such researches as those of Moreton Stillé,[1] of Peacock,[2] of Hochsinger,[3] and of Théraum,[4] we shall perhaps be able ultimately to discern them with certainty during life.

As a few points of assistance, it may be mentioned that communication of the ventricles through the septum gives rise to a systolic murmur at or near the base of the heart not propagated into the arteries, but according to Roger and to Sansom, also heard between the shoulders; that the passage of blood through an open foramen ovale very rarely engenders any sound, though presenting marked cyanosis; and that, whether coexisting with these lesions or not, the majority of instances of cardiac malformation, after the age of twelve, present signs of obstruction at the orifice of the pulmonary artery. In this instance either a systolic or a diastolic murmur may be there perceived; in the first case the second sound of the heart is weak or

[1] American Journal of the Medical Sciences, July, 1844.
[2] Treatise on Malformations of the Heart.
[3] Die Auscultation des Kindlichen Herzens, Wien, 1890.
[4] Études sur les Affections congénital du Cœur, Paris, 1895.

wanting in the second interspace on the left side. Mitral disease of congenital origin is very rare. Thrill over the præcordial region is seldom met with, except when congenital defect in the septum exists. Loud, vibratory systolic murmurs heard most distinctly over the upper third of the sternum without attending hypertrophy of the left ventricle point to persistence of the ductus Botalli. A curious result of cardiac malformation has been observed,—abscess of the brain without appreciable cause.[1]

The resemblance borne by cases of *functional disturbance of the heart*, associated with impoverished blood, to valvular affections, has already engaged our attention. The age; the anæmic look; the seat of the murmur at the base of the heart, as well as at the apex, and its soft character; the venous hum; the fact that the cardiac murmur does not entirely supersede the first sound and is followed by a distinct second sound; that the apex beat is not displaced, and that the murmur is not heard at the back, are all points upon which some stress may be laid; yet not so much as upon the absence of the phenomena of an enlarged heart. But if the question be asked, Are the latter absolute demonstrations of the existence of an affection of the valves? cannot an hypertrophied or a dilated heart, with sound valves, be combined with a condition of blood capable of producing a murmur?—we are forced to answer that such is possible. Under these circumstances, the tact of the physician may help him to a well-judged decision; but the only proof of a well-judged decision is afforded by time, or by the result of treatment that restores the blood to its normal state.

A murmur caused, in violent excitement of the heart, by *misdirection of the current*, due chiefly to temporary interference with the closure of the valves, or perhaps owing to altered tension of the valves,—causes the exact working of which I have elsewhere inquired into,[2]—may become a troublesome source of error in diagnosis, especially when heard over a heart in a state of dilated hypertrophy or of dilatation. Fortunately, a blowing sound of this origin and in this combination is comparatively rare, and we are enabled to discriminate it from an organic valvular murmur by its not being persistent. It is much more likely to be heard at the apex, or rather, according to my own observations, somewhat above the apex, than is a murmur owing to changes in the blood; and it differs from the systolic blowing sound of mitral disease partly by the peculiarity

[1] Ballet, Archives Générales de Médecine, June, 1880.

[2] On Functional Valvular Disorders, Amer. Journ. Med. Sci., July, 1869.

of seat just mentioned, partly by its non-diffusion, its usual absence at the back of the chest, the want of harshness in the inconstant murmur, and the low pitch. Murmurs of this kind are also caused by obstructive diseases of the lungs, without disease of the heart being present. They may be brought out, as John K. Mitchell has shown, by suddenly closing the hand tightly.[1]

At times a murmur is heard which is not dependent on a cardiac affection, but on *lung changes*. We find, for instance, in consolidation of the left apex, especially if the lung be also contracted, a murmur, almost invariably systolic, over the site of the pulmonary artery; or we may encounter over large cavities with thin walls, situated in the neighborhood of the heart, a systolic, cardio-pulmonary murmur, caused, most likely, by the agitation of the air in the cavity, the heart being quite sound.

These, then, are the causes which impair the value of the cardiac blowing sound as a sign of a valvular lesion. Yet they do not happen often enough to prevent us from regarding a persistent murmur as eminently indicative of an organic affection of the valves.

Let us suppose that we are convinced that the murmur is due to a structural lesion. Can we say what its precise nature is? Can we accurately foretell that the valve is merely roughened, or that it has undergone calcareous transformation, or that it has been bound down, or that it is lacerated, or that vegetations spring from it, or that its muscular attachments are sound or unsound? No, assuredly not. The most we can do is to judge whether the orifices through which the current flows are narrowed, or whether, by the valves not closing, they permit of regurgitation; and to distinguish even this we have to take into account more the time of the occurrence of the sound than its particular character or pitch. Indeed, all distinctions based entirely on either of these are not borne out by clinical experience. Valves incompetent to close the openings at which they are seated may permit a murmur to be generated of any character and of any pitch. It is true that a harsh murmur, like that of a saw or of a rasp, is for the most part occasioned by a contracted orifice with rigid valves. In obstruction at the mitral and tricuspid orifice, the murmur is mostly rough or rumbling. Broadbent[2] maintains that a loud and long murmur is significant of less structural damage and functional imperfection than a short and weak murmur.

A cardiac sound which is rare, but which, when present, is gen-

[1] Transactions of the College of Physicians of Philadelphia, 1892.

[2] Heart Disease, 1898.

erally associated with a narrowed orifice, is a distinct *musical tone* heard at the mitral or aortic valves. It resembles the cooing of a pigeon; or the auscultator listens and listens again, and directs the patient again and again to suspend his breathing, before he becomes convinced that the sound is not a sibilant râle in the lung. It is sometimes perceived merely at the beginning, or the end, or only in the middle of an ordinary murmur, and disappears and reappears. Where this rare sound is met with, the valves are usually rigid and unyielding. Yet this is not always the case. Sometimes the musical note is produced by the vibrations of clots which impede the rush of blood through the apertures of the heart, or by the loose edge of a valve flapping to and fro in the current. Occasionally, too, we hit upon it in chlorosis; but only very occasionally, and never unless it be then equally or more marked in the arterial system. We have the authority of Stokes for the observation that it may be suddenly developed and precede the signs of structural alteration of the heart. Schroetter maintains that the musical murmur is due to the vibration of a fine fibrous band stretched across the ventricle or a valvular orifice.[1]

It has been already stated that we judge best of the condition of the orifices and of the valves by ascertaining the time at which the murmur occurs. But it is also necessary to recall the state of the orifices during the movements of the healthy heart. During the contraction of the ventricles, the valves at the auriculo-ventricular openings are closed, to prevent regurgitation into the auricles, and the valves of the aorta and pulmonary artery are open. During the dilatation of the heart the reverse takes place: the valves at the origin of the great arteries are shut, and the valves which act as gates to the auriculo-ventricular apertures are swung back, to allow the stream to flow into the ventricles.

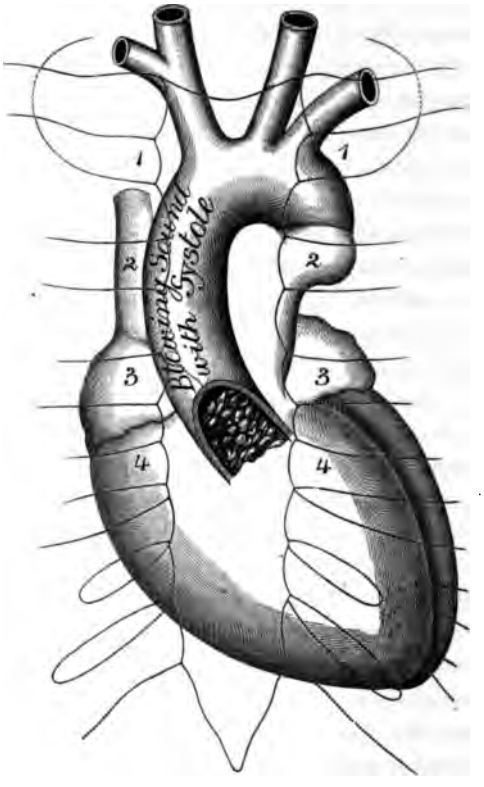
If then a murmur occur with the contraction of the heart and the first sound, it is owing to the blood either regurgitating from the ventricles into the auricles, or meeting with difficulty in passing into the aorta or pulmonary artery; if it occur after the contraction of the heart, and correspond to the second sound, it is due to the blood passing through a narrowed mitral or tricuspid orifice, or streaming back into the ventricles through incompetent aortic or pulmonary valves. But can we distinguish at which valve the mischief lies? Generally we can. By attending to the site of greatest intensity of the murmur, we become aware of the seat of its production, provided it be borne in

[1] Wien. Med. Blätter, No. 1, 1883.

mind what are the points at which to listen to the different valves. It is, however, also necessary to recollect that, as the whole heart is somewhat lowered, these points are rather below what they are in a natural state of things.

Now, we cannot always say whether more than one valve is affected. A murmur in the heart, no matter where it is generated, is usually transmitted all over the organ. If it mask the natural sounds

FIG. 48.



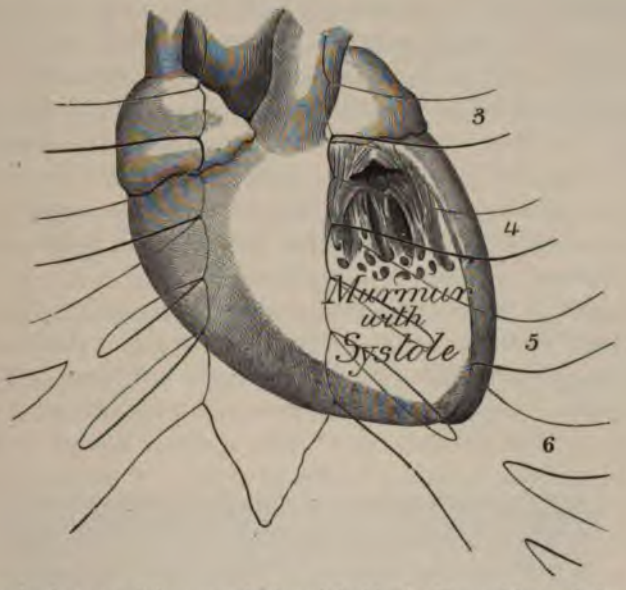
Narrowing of the aortic orifice by vegetations springing from the valves, the structure of which was, indeed, to a great extent, destroyed. The engraving illustrates also the physical signs of aortic constriction.

at other valves, it is very difficult, nay, it is often impossible, to tell positively how many of the valves are injured, unless several spots be detected at which the murmur is intense, yet not alike in character. The valves that most frequently show coexisting disease are the mitral and the aortic, particularly insufficiency of both, or aortic narrowing with mitral insufficiency. Diseases of mitral and tricuspid are also found to coexist, whether the lesion be regurgitation or narrowing.

In all instances the precise character of the murmur at the different sites of the heart is of the greatest significance.

Thus the murmur is the most conspicuous and most constant sign of a valvular lesion. The other signs and symptoms vary greatly in individual cases. Where the valves are but slightly affected, let us say slightly roughened, as they sometimes are after an attack of rheumatic endocarditis, the heart does not undergo any decided change in size;

FIG. 49.



Insufficient mitral valves permitting regurgitation of the blood. The position and time of occurrence of the most significant sign of the affection are indicated in the engraving.

the circulation is carried on regularly, and, in spite of the abnormal sound in the heart, the patient's health remains unimpaired, or it is only occasionally that he suffers from palpitations. An alteration of the valves of the heart of any extent produces, however, an alteration either in the capacity of its cavities or in the thickness of its walls, and the symptoms of dilatation or hypertrophy make their appearance along with the physical signs of extended percussion dulness and feeble or heaving impulse. Ordinarily it is the latter we meet with, because the valves of the left side are so very much more frequently diseased, and their derangements lead to hypertrophy rather than to dilatation. Affections of the *tricuspid valves* are usually connected with dilatation of the organ; hence dropsy, venous turgescence and albuminous urine are in them more especially observed; and Blakis-

ton has taught us their frequent association with engorgement of the vessels of the brain, and how this becomes the predisposing cause of cerebral apoplexy when in connection with cardiac disease. We also find in them, or rather in tricuspid insufficiency, what Mahot has more particularly called attention to,—a pulsation of the liver corresponding to each systole of the heart, perceived by gently depressing the abdominal parietes with the hand on the epigastrium. In combined tricuspid and mitral narrowing we may have the signs of pulmonary-artery regurgitation.[1] In high degrees of aortic insufficiency, a systolic apex murmur, as pointed out by Flint, is very often produced by dilatation of the mitral orifice. The murmur differs from that over the damaged aortic valves, and may be presystolic in time. In coexisting aortic and mitral insufficiency the compensatory hypertrophy is arrested. In some cases of mitral regurgitation the mitral murmur occupies only the middle or the latter part of the systole.[2] In instances of disease of three valves, as in the case reported by Shattuck,[3] double murmurs of dissimilar kind may be heard over the area of the different orifices.

All valvular lesions may be combined with pain in the præcordia, palpitations, restlessness, and disturbed dreams. And according as the deranged circulation through the heart interferes with the circulation in other parts, special symptoms show themselves prominently. Thus, we find those who labor under a *mitral disease* suffering most from cough, from dyspnœa, and from attacks of cardiac asthma, since it is the lung which has to bear the brunt of the embarrassed flow of the blood. If we examine this organ closely, the physical sounds afford direct proof of its disordered condition. Here and there are heard plentiful moist sounds from fluid which has leaked into air-tubes: here and there the respiratory murmur is roughened, and percussion shows impaired clearness. This loss of the natural resonance is at times very manifest at the upper part of the lung, and I have known it to give rise to the suspicion of tubercular deposit in cases in which the autopsy proved the pulmonary tissue to be healthy, though in a state of extreme congestion. Respiratory percussion renders the sound again clear. Mitral insufficiency generally leads to hypertrophy of the heart; mitral stenosis becomes associated with dilatation, or there is only hypertrophy of the right ventricle.

When the *aortic valves* permit of regurgitation, this gives rise to

[1] Dyce Duckworth, Clin. Soc. Transact., Jan. 1888.

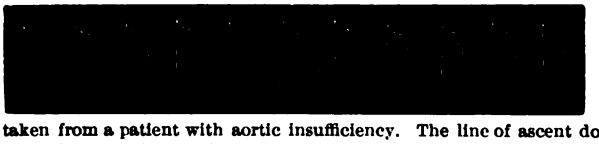
[2] Crozer Griffith, Amer. Journ. Med. Sci., Sept. 1892.

[3] Boston Medical and Surgical Journal, 1891.

effects which are perceptible along the track of the arteries. These all look superficial, and beat with apparent violence, from the force with which the thickened left ventricle is driving the blood through the tubes. The pulsation of the vessels may be even seen in the retinal vessels with the aid of the ophthalmoscope. Yet, when the finger is applied to the artery at the wrist, the strength of the beat is not so great as expected. A short, abrupt, jerking impulse is indeed communicated to the finger; but then the artery immediately recedes, proving that it was only imperfectly filled. This pulse is the only one which gives us any real information as to the state of the orifices of the heart. In general terms, it may be stated that the pulse is small and rather tense when the openings are narrowed. Still, no stress can be laid on this in a diagnostic point of view. The want of correspondence between the strength of the pulse and the force with which the heart is acting is often amazing. If the second sound can be heard in the neck over the carotid artery it shows that the regurgitation is not large in amount.[1] In marked regurgitation a capillary pulse, as seen, for instance in the finger-nails, is common.

More information than by merely feeling the pulse can be obtained by studying it with the sphygmograph. But even with this, as thus far developed, we gather in valvular diseases rather corroborative evidence than knowledge which is not attainable by other means of diag-

FIG. 50.



Sphygmogram taken from a patient with aortic insufficiency. The line of ascent does not terminate in as sharp a point, nor is the descent as sudden, as we sometimes find it.

FIG. 51.

Sphygmogram taken from a patient presenting the signs of mitral regurgitation.

nosis. Perhaps with further research the instrument may be made available to inform us with certainty of the degree of the valvular imperfection; and this would be a great step in advance. As regards the most distinctive graphical signs, we obtain them in aortic regurgitation,—a vertical line of ascent of great amplitude, a pointed sum-

[1] Broadbent, Diseases of the Heart, 1898.

mit, and a sudden descent, with comparatively little dicrotism. If there be also marked aortic obstruction, the line of ascent is oblique, or rather the first part is vertical, and following the sharp point is a gradual curve-like rise; if senile changes in the artery complicate the aortic insufficiency, the sharp-pointed process terminating the line of ascent passes into a more or less horizontal plateau. In instances of decided uncomplicated aortic obstruction there are sloping up-strokes and down-strokes.

In mitral regurgitation the pulse tracing is usually very irregular, such as is seen at times in aneurism; the line of ascent is short and unequal, and the line of descent is disposed to be oblique and to present marked dicrotism. In mitral constriction there is also, usually, irregularity; it is asserted by Mahomed[1] that the up-stroke is vertical, and that there is, especially after giving digitalis, a secondary and very characteristic contraction of the ventricle manifest in the dicrotic wave. Sansom[2] agrees in the main with this observation.

But, instead of entering into a detailed description of the pulse, however studied, or of any separate symptoms of valvular disease, let us group them together with the physical signs, according to the combination in which we are wont to meet them:

TABLE OF VALVULAR DISEASES.

SEAT OF MURMUR.	SEAT OF DISEASE.	CHARACTER OF DISEASE.	CORRELATIVE PHYSICAL SIGNS AND SYMPTOMS.
Murmur most intense at or near apex of heart.	Mitral orifice.	With impulse, means insufficiency of valves, permitting of *regurgitation*; after impulse and running into or corresponding to the second sound, or, more accurately speaking, generally preceding the first sound, presystolic, means *narrowing* of the orifice.	In mitral disease the heart very commonly undergoes dilated hypertrophy, especially the right ventricle. When there is also hypertrophy of the left ventricle, it is not simply mitral narrowing. The second sound of the pulmonary artery, heard in the second left interspace, is sharp, accentuated. The cardiac murmur is often distinctly perceived posteriorly on the left side, near the angle of the scapula. Dyspnœa and dropsy are prominent symptoms, especially dyspnœa. Cough is not unusual, and the pulse is not infrequently found to be feeble and irregular. In some forms of mitral narrowing, where the curtains are not too rigid, the murmur is always rough. This is the case usually with the presystolic murmur, which is pre-eminently regarded as the sign of mitral constriction. But in this affection all murmur may be absent, and a roughening of the first sound and doubling of the second be the

[1] Medical Times and Gazette, May, 1872.

[2] Diagnosis of Diseases of the Heart, 1892.

TABLE OF VALVULAR DISEASES. — *Continued.*

SEAT OF MURMUR.	SEAT OF DISEASE.	CHARACTER OF DISEASE.	CORRELATIVE PHYSICAL SIGNS AND SYMPTOMS.
			signs; or there may be at the apex a presystolic murmur and the second sound be lost. In mitral narrowing a thrill in the cardiac region, presystolic or diastolic, can be often felt. Mitral narrowing is frequently associated with contracted kidney.
Murmur most intense at or near the middle of the sternum, or heard with equal distinctness close to the sternum in the second interspace on the right side, and thence propagated into the arterial system.	Aortic orifice.	With impulse, means *narrowing*, or obstruction; with diastole, and taking the place of the second sound, or occurring in both sounds, the first murmur short, means *regurgitation*.	Hypertrophy of left ventricle, often to a very great degree, the compensation being very decided. All the cardiac sounds may be normal, except at the aortic valve, although they are obscured by the murmur. This is distinct in the carotids, and is sometimes as well heard at the ensiform cartilage as over the sternum and on a line with the third intercostal space, or in the third or fourth interspace near the left edge of the sternum. When the orifice is constricted, a purring thrill is frequently observed to attend the harsh or musical systolic murmur. The symptoms in aortic valve disease are often remarkably latent. There is very commonly neither dropsy nor dyspnœa. The pulse in regurgitation is abrupt and receding, and all the superficial arteries and the capillaries pulsate. It is not unusual to find a double aortic blowing sound attending aortic regurgitation, probably from slight coexisting obstruction of the orifice, though this is not always found; a double murmur is also heard in the carotids and femorals. A mitral apex murmur may be also noticeable.
Murmur most intense at or very near to the ensiform cartilage, and over the lower part of the right ventricle.	Tricuspid orifice.	With impulse, *regurgitation;* with diastole, and taking therefore the place of the second sound, or, more generally, preceding the first, *narrowing*.	Tricuspid regurgitation exists usually in combination with dilatation of the right ventricle, and therefore with the symptoms of this condition; with venous congestions, with dropsies, with difficulty in breathing. On account of the open state of the orifice, the cervical veins may pulsate during the movements of the heart; and in all cases they are distended. The pulsatile motion in the neck becomes especially visible when the breath is held in expiration. The cardiac murmur is ordinarily soft, of low pitch, is not transmitted into the arteries, and is not heard above the level of the third rib. In some cases it is so feeble as to be with difficulty discerned. In tricuspid narrowing, a very rare disease, there are presystolic murmur and thrill, cyanosis of the face and lips, great dropsy, and distention of the jugular veins, with slight, or without, pulsation.

TABLE OF VALVULAR DISEASES.—*Continued.*

SEAT OF MURMUR.	SEAT OF DISEASE.	CHARACTER OF DISEASE.	CORRELATIVE PHYSICAL SIGNS AND SYMPTOMS.
Murmur most intense at the third left costal cartilage near the sternum, or even somewhat lower, or in the second intercostal space to the left of the sternum.	Pulmonary orifice.	With impulse, is *narrowing;* taking the place of the second sound, *regurgitation.*	We have little knowledge, derived from clinical observation, of diseases of the pulmonary valves, of all the valves the ones most rarely affected. Nor does a murmur in the situation indicated, and hardly audible over the left apex or along the sternum, or in the course of the great vessels, having therefore the characteristics of a pulmonic murmur, warrant a diagnosis of disease of the valves: for it may be due to anæmia; be caused by deposits at the upper part of the left lung; or be observed immediately after or during the continuance of hemorrhage from the lungs. But these remarks scarcely hold good with reference to a diastolic murmur, and not at all as regards a double murmur. If this be present, and attended with thrill and with signs of dilated hypertrophy of the right heart, we are justified in concluding the disease to be a lesion of the pulmonary valves, or at the origin of the artery. But especially if cyanosis, continuous dyspnœa, and clubbing of the fingers exist. The murmur is not propagated into the carotids. But its position may be deceptive. We must bear in mind that in rare instances of mitral disease, especially regurgitation, the murmur is loudest at the pulmonary area, and it may be so in aortic regurgitation. Pulmonary narrowing is almost always congenital, and the systolic murmur is loud and harsh. Pulmonary insufficiency may be also congenital, or be due to malignant endocarditis.

In this manner are the symptoms and signs of valvular affections associated. But it is not exactly the combination and precisely the way in which they happen in every instance, for disorders of several valves may be conjoined.

Presuming that we have been enabled to fix accurately the state of each aperture, there is a point where all our skill invariably comes to a stand-still. We cannot tell how long it is possible for life to continue, or under what circumstances death will happen. It may take place suddenly and most unexpectedly in cases in which the amount of disease in the heart is not found to be great; and, on the other hand, life, and even a tolerable degree of health, may be maintained with valves so rigid and unyielding that the point of the knife can, at the autopsy, hardly be forced through them. In mitral disease the patient is liable to be worn out by the dropsy and the in-

creasing difficulty of breathing; and so, too, in that still more serious lesion,—tricuspid regurgitation. In affections of the aortic valves the patient suffers less, but he is more liable to sudden death.

Before dismissing these valvular affections, there are a few other matters which claim consideration, though the limits set to this work will prevent their full discussion. The blowing sound has been insisted upon as the diagnostic sign of a valvular lesion, and to insist upon this is to do no more than universal experience warrants. But there are undoubtedly instances in which no murmur reaches the ear to show that the valves are damaged.

I shall cite two examples. A man, thirty-five years of age, came under my care, complaining of palpitation of the heart, of occasional attacks of bronchitis, and of shortness of breath. His health was otherwise good. A physical examination of the chest showed the action of the heart to be extremely disturbed: the impulse was strong, and the extent of dulness in the præcordial region increased. A blowing sound was heard near the apex, but, owing to the great irregularity of the movements of the heart, it was impossible to say whether it corresponded in time to the contraction or to the relaxation of the organ. The pulse was small, frequent, and intermittent. The patient continued in this state for seven months, the beat of the heart becoming more and more tumultuous; but the murmur gradually disappeared. A peculiar clacking sound took its place, which was most distinct near the apex, and was faintly transmitted to other portions of the heart. It occurred with but one sound of the heart,—with which could not be determined. For some time before his death he had considerable cough, with a frothy expectoration and great difficulty in breathing. His face and hands had begun to swell. The immediate cause of death was pulmonary apoplexy. The heart was found in a state of dilated hypertrophy, and the mitral valves had been converted into a calcareous mass, which had left but an extremely narrow chink for the blood to pass through.

The next case presents, in several respects, a striking similarity. A gentleman, about fifty years of age, who had led a gay and somewhat dissipated life, noticed that he experienced difficulty in breathing on the slightest exertion. An inquiry into the state of the heart furnished a clue to the dyspnœa. The size of the organ was evidently increased, and its rhythm very irregular. The impulse was strong; but the sounds were normal, except near the apex, where, taking the place of one, was heard a dull but very marked clack. When the hand was applied over this point, it felt a vibration of very much the same character as that which the ear could hear, and, like

this, it was only distinctly perceptible at or near the apex of the organ. The diagnosis of disease of the mitral valves was made, and it proved to be correct. The dyspnœa became greater and greater; the feet, and subsequently the abdomen, were distended with fluid; and the patient died with all the symptoms of an unmistakable valvular lesion.

I might cite more such cases; but these two present the main features of all. All the instances of valvular disease I have met with, unaccompanied by blowing sounds, have been instances of disease at the mitral orifice, and of extreme narrowing of that orifice. They were all attended with excessive irregularity of the action of the heart, and with hypertrophy. They all produced difficulty of breathing. They all presented the peculiar clacking sound most marked near the apex. In some, another sound, more like that heard in health, followed it; in others, not. In some, the blowing sound gradually disappeared; in others, none was perceived when first examined; and in others, again, it could be caught occasionally, as a very short whiff, along with the clacking sound. In all, the impulse was strong and very variable in its rhythm, and a peculiar movement was felt near the seat of the apex,—not the purring tremor that so commonly accompanies the movements of a heart the valves of which are damaged, but a more localized vibration, similar to the sound the ear hears.

These cases are probably of the same nature as those that are every now and then reported as valvular lesions in which the sounds of the heart were normal. I cannot think that with a disease of the valves they ever are so. There may be no blowing sounds present, but the sounds of the valve affected must be different from what they are in health; and it may be said again, in all truth, that to hear the natural sounds of the heart well defined is to be able to exclude a valvular disease.

Valvular disease may be at times suddenly developed, from *rupture of a valvulet or of a papillary muscle* by a severe strain. I have known such cases to happen where there was nothing in the history to lead to the belief of previous disease, though often there is some preceding disorganization, such as a granular or a fatty change. One of the most striking diagnostic features is the quickly developed organic murmur attending the signs of disordered circulation and cardiac distress; another, the occurrence of pain in the region of the heart. Rupture may happen in the affected valve of an ulcerative endocarditis without any extraordinary strain. The previous history, the sudden aggravation of the cardiac symptoms, will furnish an explanation of the accident.

Let me also here briefly discuss another question,—whether the valvular affection shows any signs by which we can recognize it *before the development of a murmur*. We cannot do so with any certainty; although marked alteration—such as dulness of sound confined to or most obvious at a particular valve, the signs of preceding or of growing hypertrophy, and, where the aortic valves are concerned, a distinct accentuation of the second sound, while the first has become dull and changed—might make us suspect what is about to happen. A doubling of either the first or the second sound, especially of the latter, is often, according to the observations of Sansom, an early sign of the development of mitral narrowing. Gibson[1] dwells on the doubling of the second pulmonary sound in mitral obstruction at any stage, and on the great tendency to the appearance and disappearance of the doubling; while Broadbent[2] maintains that a mitral systolic murmur which is retarded, following the first sound at a brief interval, shows that the changes in the valve are slight.

Displacements of the Heart.

The heart is a very movable organ. Its apex is tilted upward by an enlarged liver, by an abdominal tumor, or by a pericardial effusion. It gravitates towards the median line when the walls of the heart have increased in weight and firmness. But these changes are hardly of a nature to attract as much attention as finding a heart beating on the right side of the sternum.

Now, it is not very uncommon to meet with it there; and the question immediately arises, What does this strange alteration in its situation signify, and how is it brought about? It is usually produced by pressure exercised on the heart by accumulations of fluid or of air in the left pleural cavity, and therefore denotes, as a rule, a pleuritic effusion or a pneumothorax of the left side, and is accompanied by distention of that side. In rarer instances, the heart is pushed across by a highly distended emphysematous lung; in still rarer instances, it is drawn over to the right side by a shrinking of the lung, attended with dilatation of the bronchial tubes, the so-called pulmonary cirrhosis. It is sometimes found on the right side, because it has been forced there by a pleuritic effusion and has formed adhesions, and when the fluid was absorbed it was unable to return to its natural place. In this case the left side will be markedly retracted, and not the right, as it is if cirrhosis of the right lung be the cause of the abnormal position of the heart.

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[1] Diseases of the Heart, 1898.

[2] Heart Disease, 1898.

The displacement may further have been brought about by a cancerous or an aneurismal tumor, or by any of the abdominal viscera having slipped into the chest through a hernial opening in the diaphragm; or it may be congenital. But these all are causes which seldom exist. Pratically speaking, transpositions of the heart are met with in conection with diseases of the lungs. We shall merely add that a congenital displacement cannot be diagnosticated unless all other causes capable of producing a displacement have been proved to be absent; and that a dislocated heart is able to perform all its functions. It may even be attacked by acute disease; the recognition of which,[1] under such circumstances, belongs to the triumphs of physical diagnosis.

SECTION III.

THORACIC ANEURISM.

An aneurism of the aorta, whether caused by a disease of the coats of the artery or not, whether true or false, may affect any part of the vessel. Yet it is chiefly at the ascending portion and at the arch that it is met with. When it occurs just after the artery has left the heart, it is prone to elude discovery. Higher up, nearer to or at the arch, it more rarely escapes detection. The tumor manifests itself by a local bulging, varying in extent and situation according to the extent and situation of the aneurism. A single rib alone may be raised, or nothing but a fulness may be observed. But some prominent spot is generally detected, and when this is percussed it is more resistant, and returns a duller sound, than normal. Yet neither the bulging nor the dulness on percussion is of as much significance as finding a distinct pulsation remote from the beat of the heart. Every time the latter is perceived, an impulse is communicated to the finger at the point in the chest walls which appears to project; that is, usually on the right side of the sternum in the second intercostal space, or in the same interspace on the left side, or immediately under the top of the bone. Occasionally the beat is double, at times so violent as to shake the head of the listener, and almost always, unless the aneurism be filled with solid clots, stronger than the beat of the heart.

The impulse may be accompanied by a distinct thrill. But this is not always present, and, when present, is not always constant, since it may disappear and reappear. It is thus a serious mistake to regard the thrill as the requisite sign of an aneurismal enlargement; yet

[1] As by Stokes. See Diseases of the Heart, p. 463.

there is no mistake more common, except, perhaps, one,—to consider that the motion of the blood in the sac must necessarily engender a murmur. The ear, applied over the prominence, hears often nothing that in the least resembles a murmur, but sounds like those of the heart, sometimes two, the first weighty and prolonged; sometimes but one, and that one longer and more intense than the corresponding first sound over the ventricles.

Thus, then, neither thrill nor murmur is essential to the diagnosis of an aneurism. What is much more essential is to find two points of pulsation in the chest,—two hearts, apparently, each with its own distinct beat, its own distinct sounds.

An aneurismal tumor in the chest gives rise to symptoms which vary somewhat according to its seat and size. Prominent among them stand those occasioned by pressure. The sac presses on the adjacent air-tubes, and shortness of breathing, or peculiar cough and signs counterfeiting those of a chronic laryngeal disease, are the result; or it presses on the œsophagus, and the patient suffers from difficulty in swallowing; or it presses on the subclavian artery, and the pulses at the two wrists are noticed to be strikingly different both in volume and in time; or on the carotid, and pain in the head, dulness of mind, occasional giddiness, and flashes of light before the eyes are complained of; or on the venous trunks, and the superficial veins of the neck and thorax are seen to be engorged, and the skin becomes very puffy and swollen; or on the trunk of the sympathetic nerve or on its ganglia and their communications, and marked contraction, or, in rarer instances, dilatation, of the pupil of the eye on the side of the aneurismal swelling, is perceived, or unilateral sweating becomes an annoying complication. All these signs, then, denote pressure, and pressure connected with a pulsating tumor in the chest means an aneurism.

I say with a pulsating tumor, because a cancerous or other *intra-thoracic morbid growth* may produce exactly the same signs of compression as aneurismal tumor,—the same stridor, the same cough, the same feebleness of respiration in one lung from partial obliteration of its bronchial tube. But the solid tumor, large though it be, does not pulsate, or if it do, pulsates but very feebly, and not with the heaving motion of a distending aneurismal sac. The tumor, which for the most part has its seat in the mediastinum, renders a large surface dull on percussion, and communicates a much greater feeling of resistance to the percussing finger. Yet the ear listens in vain over the prominence for the weighty sound with each beat of the heart, or for the hoarse murmur of the blood streaming through the

sac. It is only where a solid growth presses on the artery that any murmur is perceived, and there is always a distinct second aortic sound. Further, a tumor is not confined to the course of the aorta; it is more commonly connected with a distended state of the veins of the neck and thorax, and with œdema of the arm and chest; the pain it occasions is often more continued, and less neuralgic in its nature, and the dyspnœa is not infrequently paroxysmal. Moreover, as most thoracic tumors are cancerous, the violent constitutional disturbance, the formation of external swellings, the enlargement of the glands in the axilla and the neck, and the peculiar currant-jelly expectoration, aid us in arriving at a correct conclusion. Sarcoma, lymphomata, and lymphadenomata of the mediastinum come next in frequency to cancer.[1] They all tend to grow inward rather than outward, and affect the anterior mediastinum far oftener than the other two spaces. Then the history is of some value in the diagnosis. In aneurism it points to gout, to aortitis, to alcoholism, to syphilis, to strain, to an embolic infarct, to infectious arteritis from mycotic invasion of the aorta, and there is generally some evidence of arterial degeneration. In tumors of the mediastinum, Wintrich's tracheal sound, the sound which issues from the trachea on percussing the chest, can be elicited by moderate percussion over the manubrium during the act of inspiration. The same may be, however, also found in aneurism of the ascending arch of the aorta.[2]

The most difficult diagnosis—it is often an impossible one—is between an aneurism filled with solid clots and a tumor. The history of the case is here of the greatest importance; and there is generally less pain in these altered aneurisms than in tumor. The physical signs will not help us. As a rule, it may be said that we do not meet in the latter with the ringing second aortic sound, or the shock with this, which happens in aneurism,—happens mostly even when it is filled with clots.

As regards *abscess of the mediastinum*, we do not find the pressure signs generally so marked as in aneurism, and we may be able to detect fluctuation at the edge of the sternum or at the suprasternal notch. The pain is usually very great; the elevation of temperature is significant. The sounds over the mass are not those of an aneurismal sac; there are certainly no distinctive murmurs, and we find no marked expansile pulsations. This absence of distinct pulsation was the main point of dissimilarity between an aneurism and an ab-

[1] Hare, Mediastinal Disease, 1889.

[2] C. F. Hoover, Amer. Journ. Med. Sci., Oct. 1899.

scess of the mediastinum in a case some time since under my care, which, after lasting a year, and simulating aneurism most closely in the pain, the dulness on percussion, the difficulty of breathing and of swallowing and the altered voice,—having, therefore, pressure signs much more marked than usual,—got well by the abscess breaking internally and the discharge, as expectoration, of large amounts of purulent matter. In inflammatory thickening of the mediastinum the impulse of the heart is weak and the sounds are faint.[1]

The obvious inequality of the pupils, which is found in a certain number of cases among the signs of an aneurism, is of little aid in a differential diagnosis from intrathoracic tumor, for a thoracic cancer has been noted to occasion the same.[2] The rarity of a non-aneurismal tumor in the chest is, however, very great; and, practically speaking, when the signs of intrathoracic tumor are met with we shall be generally correct in thinking that it is an aneurism we have to treat, even should the pulsations not be very obvious.

The sphygmograph will at times aid us in the diagnosis of an aneurism, though its value is not on the whole great. Its most distinct significance is in showing clearly the difference between the two pulses. Of one radial the sphygmogram may be normal; the other tracing furnishes a characteristic record,—a sloping up-stroke, a rounded apex, an obliteration of the secondary curves.

Another sign of aneurism which has been much studied, and especially by MacDonnell,[3] is the so-called tracheal tugging. To obtain it the cricoid cartilage is firmly grasped and the trachea put on the stretch by pressing upward. If an aneurism is adherent to it or near it, a significant tugging will be felt. In deep-seated aneurisms this sign is of special value; particularly significant is it of aneurism of the transverse portion of the arch. But it is not absolutely characteristic of aneurism. It has been found by Grimsdale[4] and by Ewart[5] in other conditions, and even in healthy subjects.

Let us suppose that we are satisfied, owing to a marked impulse, that we have not a solid growth or an abscess to deal with, can we say that it is an aneurismal enlargement? If there be also swelling and signs of pressure, we can; should these not exist, we cannot be so sure. For a pulsation in the chest not immediately over the region

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[1] Wilson Fox, Treatise on Diseases of the Lungs.

[2] MacDonnell, Montreal Medical Chronicle, June, 1858; Gairdner, Clinical Medicine, and Ogle, Medico-Chirurgical Transactions, vol. xli.

[3] Lancet, March, 1891.

[4] Practitioner, London, Feb. 1892.

[5] British Medical Journal, March, 1892.

of the heart may be owing to other causes. Where the aortic valves are insufficient, there may be a pulsation in the aorta; an empyema may pulsate; a dilated auricle may occasion an impulse separate from that of the ventricles; a pulmonary artery surrounded by consolidated lung may distinctly exhibit its beat. In all of these the signs of pressure on the surrounding parts are wanting; and, on the other hand, they show phenomena which an aneurism lacks.

Insufficient aortic valves are accompanied by hypertrophy of the left ventricle. So is at times a thoracic aneurism; but, instead of the throbbing at the upper anterior part of the chest being attended, as in aneurismal swelling, with a natural or with an unequal beat at the wrist, there, as well as in the larger trunks in the neck and arms, is perceived that strong and peculiar pulsation so characteristic of inadequate aortic valves. Then, again, a murmur is invariable in this affection, and is usually a loud double murmur, most distinct at the right base of the heart, and associated with a double murmur in the femorals made evident by pressure with the stethoscope. This is very rare in aneurism of the aorta; moreover, the murmur heard over an aneurismal pulsation is better marked over its seat than over the heart, and is mostly single, short, hoarse and of low pitch, systolic, only in very rare instances diastolic. It differs in distinctness as well as in quality from the murmur discerned at the base of the heart, which is transmitted from the aneurism, or depends upon coexisting cardiac disease. Then the sphygmographic tracings may be also of value. Those of aortic regurgitation are characteristic; while an oblique line of ascent, a loss of the summit wave, and a modification of the dicrotism are usual when an aneurism is seated on a main trunk after its origin from the aorta. When the aorta is dilated, as well as its valves diseased, the diagnosis as regards aneurism is much more difficult. But even then we lack the distinct throbbing, the signs of pressure, and the unequal pulses.

Coarctation or constriction of the aorta, which in rare cases is associated with the valvular affection, may be here mentioned. It generally happens just at or below the insertion of the ductus arteriosus, and furnishes as its only special signs a dilatation of certain collateral vessels at the upper part of the thorax, and feeble, retarded pulsation of the femorals. The arteries of the head and neck, as well as the epigastric and mammary arteries, throb, and there may be a marked thrill at the upper part of the chest near the sternum, and a murmur there louder than over the heart; but pressure signs are absent, and the dilated vessels are often the seat of a purring noise.

A *pulsating empyema* is seldom met with; yet a collection of fluid

in the cavity of the chest may vibrate with the motion of the heart, and throb with such distinctness as closely to simulate an aneurism. To determine the real nature of the pulsation in these cases, we must attach importance to the situation of the expanding mass, which is not often that of an aneurism, and to the signs which point out that liquid has accumulated within the pleural sac. We also note the circumstance that over the seat of impulse there are no peculiarly marked sounds, no murmurs, no thrill; moreover, the beat, which is wide-spread, is not apt to be so strong as that of the heart, which is displaced. The pulsation may happen both in acute and chronic pleurisy, and be associated, as in Osler's[1] case, with persistent tenderness of the thoracic walls. There may be a number of these pulsating tumors.[2] Pulsating pleurisies are nearly always left-sided and purulent; there is generally latent pneumothorax present.[3] In one of Wilson's cases[4] the pulsation disappeared immediately after aspiration.

A *dilated auricle*, the walls of which are at the same time hypertrophied, may give rise to a movement separate from that of the beat of the ventricle. Bouillaud cites an example of this nature, in which a double motion was perceptible in the second intercostal space of the left side, in a person whose heart was extensively hypertrophied and whose mitral valves were indurated. Such cases are extremely rare. The signs of an accompanying valvular affection and of enlargement of the ventricles, and the probable presence of dropsy, would serve to distinguish a dilated auricle from aneurism of the arch. And this is the only form of enlargement of the heart which is likely to be mistaken for an aneurism. In cases of hypertrophy or dilatation as we ordinarily meet with them, there is but one motion discernible,—that over the ventricles,—and not two beats at some distance from each other; the signs of pressure, too, are wanting. In dilatation of the right auricle, Sansom notes a vibration to the right of the sternum and a wedge-shaped line of dulness joining the dulness of the liver.

A *pulmonary artery* surrounded by consolidated lung-tissue may cause—especially if the vessel be somewhat widened—a distinct pulsation. But the seat of the dulness near the apex of the left lung; its non-extension over the median line; the limitation of the murmur to the site of the pulmonary artery, or, in some instances, to this vessel and the subclavian; the sharply-defined second sound of the pul-

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[1] Amer. Journ. Med. Sci., Jan. 1889.

[2] Henry, Proceedings of the Philadelphia County Medical Society, vol. iii.

[3] Comby, Arch. Gén. de Méd., April, 1889.

[4] Transactions of the Association of American Physicians, 1893.

monary artery in the second interspace on the left side; the symptoms and physical signs of phthisis, the most common cause of the consolidation, and a morbid condition which of itself would appear to exclude an aneurism; the absence of pain and of the phenomena caused by pressure,—all these prove the murmur and the pulsation not to be due to an aortic aneurism. Absence of pain and of pressure signs, and accentuation of the second sound, are also the chief signs by which we distinguish those comparatively rare cases of murmur in the second interspace, close to the left of the sternum, which are due to retraction of the lung and uncovering of the heart and pulmonary artery. The murmur, which has been specially studied by Quincke[1] and Balfour,[2] is systolic and loud, and mostly disappears on deep inspiration. The pulsation is marked, though not so strong as that of the heart; the singular murmur is supposed to be owing to compression of the pulmonary artery by the heart during the systole. In many respects it is like the murmur, which I have elsewhere investigated,[3] heard over the pulmonary artery in certain lung affections.

Another abnormal condition which may be mistaken for an aneurism is a *malformation of the chest*, particularly when produced by great prominence of the upper part of the sternum. This error is more especially apt to occur if there be coexisting disturbance of the heart, whether of functional or of organic origin. I have seen cases where the beating of the arteries of the neck, accompanied by an enlargement of the thyroid gland and by cardiac palpitation, was believed to be an aneurism, mainly because it was combined with very decided prominence of the upper portion of the sternum. But there were no distinctly localized tumefaction and pulsation, no altered sounds, no signs of pressure. I have also met with instances in which the active pulsation of the thyroid gland, both in exophthalmic and in ordinary goitre, gave rise to the idea of an aneurism, but in which no change of the chest walls existed. In such cases the carotids and radials beat equally; a blowing murmur, attended by a continuous hum, is heard—certainly in instances of exophthalmic goitre—over the enlarged gland; there is nowhere a point of localized pulsation, and there are no signs of pressure.

Malposition of the aorta, due to rickets, may simulate an aneurism closely. Balfour[4] has pointed out how misleading may be the abnor-

[1] Berliner klinische Wochenschrift, 1870.

[2] Lectures on Diseases of the Heart, London, 1876.

[3] Amer. Journ. Med. Sci., Jan. 1859.

[4] Diseases of the Heart, London, 1876.

mal pulsation with the dulness on percussion, and the right-sided prominence of the chest. Moreover, thrill, murmurs loudest over the pulsating mass, and cardiac hypertrophy, may coexist. We must be guided in our opinion by the history of the case; by the distortion of the spine; by the extended superficial dulness on percussion, out of proportion to the extent and strength of the pulsation of the tumor, which is less forcible than that of the heart; by the displaced position of the heart, which is tilted upward and thrown over more to the right; and especially by the absence of any signs of pressure.

The signs of pressure play, then, a very important part in the diagnosis of an aneurism. They are rarely wanting, although they do not always manifest themselves in the same manner: sometimes it is bone, sometimes lung, sometimes œsophagus, sometimes nerve-fibre, which bears the brunt of the distending swelling. These signs of pressure are not present if the sac be very small; or not prominent, if the artery be simply dilated, in which case nothing but a constantly pulsating tumor can be detected. At times evidences of compression may be recognized when no throbbing swelling can be discerned, and from them the true nature of the case inferred. Whenever, indeed, obstinate and anomalous thoracic symptoms, which might be explained by the presence of an aneurismal sac, occur in a person whose lungs and heart appear to be in every respect sound and whose general health is not materially affected, we may suspect an aneurism to be the source of the disorder. So, too, if there be strange laryngeal or œsophageal manifestations.

The symptoms of *chronic laryngitis* especially are at times astonishingly simulated, and it may happen that the patient, trusting to his feelings, refers obstinately to the chest as the seat of the disorder, while the physician as obstinately sees nothing but the presumed affection of the larynx. There is, as in chronic laryngeal disease, alteration of the voice with cough; but the voice frequently retains much of its natural character. Hoarse it may be, but, as the pressure varies, it alters rapidly both in pitch and in power. The cough is commonly paroxysmal, and has a ringing sound. Dyspnœa is a constant symptom. It is often attended with wheezing or stridulous breathing, which is not persistent, and is sometimes produced only after a deep inspiration. The stridor, however, as Stokes points out, differs from that of an obstructive disease of the larynx by its seeming to issue from the notch at the sternum, and not from above, from the larynx itself. If, in addition, the respiration be found to be markedly unequal in the two lungs, the diagnosis of aneurism may be ventured upon; and it will be confirmed by finding no change in the larynx sufficient

to account for the laryngeal symptoms, or such a change—paralysis of only one cord, for instance, or paralysis of an abductor on one side—as could be readily explained by pressure on one recurrent nerve.[1] Of course, the detection of dulness on percussion, of sounds stronger than or otherwise different from those in the cardiac region, or the occurrence of a hemorrhage, would place the diagnosis beyond doubt. A systolic sound or thud in the brachial artery is also a sign to which importance may be attached.[2]

In some cases of aneurism, pain is among the earliest symptoms, and the patient complains much of it before there is a single physical sign indicative of the presence of a tumor. The pain is dependent upon pressure on the nervous filaments: it may shoot towards the shoulder or the neck, along the arm, or deep into the centre of the chest. Dull, deep pain, boring and constant, occurs when the pressure of the sac is leading to absorption of the vertebræ. Over the seat of the swelling there is often pain, with great tenderness.

The severity of the pain may give rise to emaciation and fatal exhaustion; but usually the patient's life is cut short by the aneurism bursting, either externally or into the trachea, bronchial tubes, œsophagus, pericardium, pleura, pulmonary artery, or spinal canal. Yet it is not always the first rent which leads to the fatal issue; this, when the aneurism breaks externally, may not happen for weeks after the accident.[3]

Now, can we foretell the course of an aneurism, and the probable mode of death it is likely to occasion? We cannot; since we cannot determine accurately its seat nor know what tissues are likely to be encroached upon. It is true that, when the swelling gives rise to phenomena like those of angina pectoris, we may surmise it to be in the ascending portion of the aorta and near the cardiac plexus of nerves, and look for its breaking into the pericardium or the pulmonary artery; when it is accompanied by laryngeal stridor or other laryngeal symptoms, it probably involves the posterior and lower portions of the arch, and will cause death by strangulation or by exhaustion; when it produces much dyspnœa, it is apt to be seated in the descending part of the arch, and death may take place by the aneurism bursting into a bronchial tube, or by pneumonia. But in regard to all these matters we can do little else than conjecture.

[1] In the chapter on Diseases of the Larynx, the forms of laryngeal palsy from an aneurism have been more specially examined into.

[2] Glascow, New York Medical Journal, Sept. 1894.

[3] Webb, Amer. Journ. Med. Sci., Oct. 1874.

PLATE III.

Aneurism of the ascending aorta on the right side, springing from the base of the heart. The shadow of the heart is projected into the lower part of the picture. Skiagraph by Dr. Leonard.

An aneurism of the *descending aorta*, between the arch and the diaphragm, produces, if extensive, dulness on percussion and bulging posteriorly, and may exhibit the same physical signs and symptoms as an aneurism in the neighborhood of the arch. A gnawing sensation in the vertebræ has been especially noticed; so have difficulty in swallowing and stridor on the left side of the chest. Yet, in spite of the most careful scrutiny, an aneurism of the descending aorta often escapes detection, or its physical signs, as a case recorded by Walshe[1] proves, may exist to the right instead of to the left of the spinal column, because the vessel has been dragged across the median line by its enlargement. In aneurism of the descending thoracic aorta near the diaphragm, we have expansile pulsation, but not stridor; there are, as Gibson[2] points out, marked signs of compression with vesiculo-bronchial or bronchial breathing, and increased vocal resonance at the lower part of the left lung. In aneurisms of the descending aorta, perhaps even more than in aneurisms of other portions of the aorta, we get the greatest help from the Roentgen rays, and cases that cannot be otherwise recognized can thus be diagnosticated.

An *aneurism of the heart* may in exceptional instances produce localized bulging in the cardiac region. But, whether it does so or not, it is beyond the reach of positive diagnosis. We may suspect it if the bulging have been preceded by signs of fibroid degeneration of the walls of the heart. Obstructed coronaries producing the myocardial changes are its most common cause. Pericarditis with adhesions near the aneurism has been also noticed. In a number of instances we have a syphilitic history.

In rare instances we find a varicose aneurism communicating with either the ascending or the descending vena cava. These aneurisms mostly present the ordinary signs of a thoracic aneurism; but, in addition, great venous enlargement above the diaphragm, with œdema of the face and hands and arms; a purple hue of the face and the upper part of the body, and spots of ecchymosis in the skin; a jerking pulse; a purring thrill; and a whirring systolic murmur,[3] diffused all over the front of the chest. The œdema and the symptoms of venous disturbance come on suddenly. In occlusion of the vena cava the great venous distention is not accompanied by the physical signs of aneurism, nor by thrill, nor by cyanosis and œdematous swelling.[4]

[1] Diseases of the Heart.

[2] Diseases of the Heart and Aorta, 1898.

[3] As in Mayne's case, Dublin Quart. Journ. of Med. Sci., Nov. 1853; also in Glascow's case, St. Louis Courier of Med., Jan. 1885.

[4] Arthur V. Meigs's case, Transact. Coll. of Phys. of Phila., 1886.

Let us, in conclusion, glance at the other kinds of aneurism within the thorax,—that of the innominate and that of the pulmonary artery.

An aneurism of the *innominate artery* is strictly limited to the right side of the body. It differs from that of the arch by the higher situation of the pulsating swelling; by the displacement of the clavicle; by the comparative absence of signs of pressure on the larynx and œsophagus; and by the fact that compression of the right subclavian and carotid diminishes the beat of the tumor, while it exerts no effect on an aortic aneurism. Such are, at all events, the marks of distinction indicated by the observations in Holland's[1] excellent memoir. An additional sign is mentioned by Wardrop.[2] It is that when the innominate is affected, the difficulty will appear first on the tracheal side of the sterno-mastoid; but on the cervical side, if the aneurism be of the subclavian. In aneurism of the innominate, further, as the tumor is under the right sterno-articular articulation, percussion does not detect any distinct enlargement of the arch of the aorta.

An aneurism of the *pulmonary artery* is a rare disease. Its main phenomena are: a strongly pulsating swelling, perceptible to the left of the sternum, and limited to the second intercostal space; a marked thrill with each expansion of the aneurism; and in some instances a rough murmur, which is not discovered at the notch of the sternum or above the clavicles; lividity of the face; dropsy; great difficulty of breathing; and the absence of obvious evidences of pressure.[3] The situation, too, of the physical signs is important; yet an aneurism of the arch may occasion a pulsating tumor mainly to the left of the sternum, and may even break into the pulmonary artery. A mere distinct beating of the pulmonary artery is discriminated from an aneurism of this vessel by the non-existence of a palpable swelling, of dropsy, of embarrassed breathing, of lividity of the face, and by the usually co-existing signs of some consolidation of the left lung.

Occasionally we meet under the outer half of the left clavicle with a pulsating tumor presenting thrill and murmur, and dilated veins above. The signs often suddenly disappear. These "mimic" or phantom aneurisms[4] are apt to come back after excitement and after movement of the arms. They are thought to be due to temporary dilatation of the artery from vasomotor paralysis, limited to a large vessel or to part of it.

[1] Dublin Quarterly Journal, vol. xii.

[2] Holmes's Surgery, vol. iii. p. 562.

[3] In the case detailed by Skoda, Auscultation and Percussion, the dropsy was great, and the face cyanotic; there was no murmur over the pulmonary artery.

[4] See paper by Samuel West, St. Barthol. Hosp. Rep., 1880.

CHAPTER V.

DISEASES OF THE MOUTH, PHARYNX, AND ŒSOPHAGUS.

THE diseases of this part of the digestive system need not here be described at any length, because many of them have been already considered. Yet some require further examination.

MOUTH.

Soreness of the mouth, pain in masticating, and a fetid breath are often complained of in diseases of the oral cavity. Let us suppose a patient to present himself with such symptoms. The interior of the mouth is exposed to a strong light, and its different parts are inspected.

The gums are noticed to be swollen and injected, and the mucous membrane lining the cheeks reddened.—This is a state of things observed in the different forms of *stomatitis*. In the *common diffused inflammation*, the result of direct irritation, such as the swallowing of hot liquids or corrosive substances, or an accompaniment and consequence of gastric disorder, the redness is marked; any attempt at chewing is painful; the taste is impaired; a flow of saliva takes place from the mouth, and superficial ulcerations occur at its various parts. In *mercurial stomatitis* there are much the same symptoms; but the more copious discharge of saliva, the pain in the jaws, the spongy gums, the loosening of the teeth, the enlarged tongue, exhibiting their impress, the painful and swollen state of the salivary glands, and the peculiar nauseous breath, testify to the specific character of the inflammation. Ptyalism may be accompanied by ulceration of the lips or cheeks, and followed by caries or necrosis of the bones of the jaw. The sore mouth of *scurvy* is distinguished from either of the preceding forms by the spongy, purplish, or livid gums, which bleed on the slightest touch, by the eruption or ecchymoses on the skin, and by the other signs which attend a scorbutic state.

The gums and the inside of the cheeks and lips are covered with a whitish curd-like exudation.—This constitutes the form of stomatitis known as *thrush*, so frequent in infants at the breast, and so constantly associated with intestinal disorder, with diarrhœa, with colicky pains, and with a feverish skin and a hot, dry mouth. Very similar

to it, regarded indeed by some as identical, is the aphthous ulceration, to which adults as well as children are liable. Here, too, a whitish deposit is perceived in various parts of the mouth; it is apt also to be combined with thirst and with gastric or intestinal disturbance, and the breath has a very disagreeable odor. The recognized difference consists in the presence of the superficial or shallow ulcers which may be detected when the white crusts that cover them are removed, and the vesicular nature of the disease during its formative stage. Then more or less redness surrounds each spot, the ulcers are slightly raised at their borders, bleed easily on pressure, and may be irregular from several running together; their grayish covering is soluble in ether, and presents many oil-globules under the microscope. On the other hand, the microscope shows us in thrush a special parasitic formation, the oidium, or mycoderma, albicans.

Ulcerations are perceived on the gums, tongue, and various parts of the mouth.—We meet with ulcers in the ordinary, in the mercurial, in the scorbutic, and in the aphthous inflammation of the mouth. They are also seen attending the well-known "sore mouth" of pregnant women, and accompanying tuberculosis. But ulceration is apt to exhibit its most horrible features in the sore mouth of syphilis, and in that essentially ulcerative disease called *cancrum oris*, or gangrenous stomatitis. In the former the fauces as well as the mouth are, as a rule, involved, and the ulcers show peculiarities which we shall presently study. The latter is an affection which prevails especially in enfeebled constitutions. It is seen chiefly in hospitals, and not uncommonly in epidemics. It begins with pain in the gums, and these soon swell, redden, and bleed readily. They are covered with a soft, grayish exudation, which often extends to the soft palate. If the layer of exudation be scraped away, a bleeding, ulcerated mucous membrane comes into view. The breath is most offensive; a profuse flow of saliva is noticeable; perforation of the cheek quickly takes place; the bones may be laid bare, the teeth loosened; there is usually fever, often of hectic type; yet the disease does not uniformly progress with activity; it may last for weeks. *Tubercular ulceration* is distinguished usually by a chronic course and by the presence of tubercle bacilli in the granulations and in the submucous tissues.

The tongue is red and swollen.—Changes in color and in appearance of the tongue occur in general diseases of the system, and more especially in those of the alimentary canal. The tongue is also more or less involved, at all events its mucous membrane is, in the different forms of stomatitis. An abnormal state of the covering of the tongue is, therefore, far from being a sign that the organ itself is primarily affected.

Occasionally, however, we do meet with affections of its deeper structures. Its nerves may be the seat of violent neuralgia; its muscles may be paralyzed; it may become hypertrophied or cancerous; it may undergo progressive atrophy; or it may be in a state of *acute inflammation*. The latter is, perhaps, the most frequent of its maladies, and is readily recognized by the red, swollen look of the organ, joined to a burning pain in it, and either to great dryness of the mouth or to constant dribbling. The swelling usually begins at the anterior portion, and may become so considerable as to threaten suffocation; the inflamed tongue fills up the fauces and protrudes out of the mouth, and the unhappy patient can neither swallow nor utter a word. He has active fever, headache, great restlessness, and intense thirst,—symptoms which last for several days, and until the inflammation subsides. This may run on to suppuration or gangrene; in some instances it leaves a permanent induration that may be mistaken for a cancerous nodule. Acute glossitis is a dangerous complaint; fortunately, it is a rare one. Its most frequent cause, as now seen, is direct injury, either from wounds or the stings of venomous insects, or from the introduction of corrosive substances into the mouth. Its most frequent cause formerly was the abuse of mercury pushed to salivation. At times it is observed as a complication of scarlatina or of erysipelas.

Other affections of the tongue connected with diseases of its structure have been mentioned in the first part of this volume. *Cancer* of the tongue produces the greatest alteration in the form and texture of the organ. *Syphilis* of the tongue gives rise to deep fissures, ulcers, or mucous patches and gummous nodules which may be difficult to distinguish from cancer, except by the history and the absence of pain. As a sign of recovery from syphilis, the tongue may present a peculiar indented appearance, similar to what is seen in the syphilitic liver.

FAUCES.

The throat, or fauces,—that is, the parts at the back of the mouth which are brought into view when the lips are widely opened, such as the half-arches, the uvula, the tonsils, the posterior wall of the pharynx,—may be involved in the same diseases as the parts situated in front. The contiguity of these structures is in fact such that any morbid action is apt to spread to them, or to extend from them either forward or downward into the pharynx, and even into the larynx. The most common affections of the fauces are inflammation and ulceration, both of which occasion a feeling of uneasiness in the throat, and also difficulty or pain in deglutition, and both of which are readily enough detected by the attendant changes in color, swelling, or exudation.

In the ordinary inflammation of the fauces, the *simple angina*, or sore throat, the parts are of a bright-red color, and the uvula is long and swollen, and by dropping on the tongue gives rise to a constant disposition to swallow, although the act of swallowing is attended with pain. Associated with the angina are coryza and febrile disturbance; and, owing to the inflammation travelling up the Eustachian tube, the sense of hearing is impaired.

Tonsillitis.—When the inflammation penetrates the substance of the tonsils, as in *quinsy*, much the same general symptoms occur as in ordinary angina. But the sense of constriction in the throat is greater: so is the difficulty in swallowing; and liquids are apt to return through the nose. The voice is thick, and has often a peculiar sound; it is painful to the patient to talk, and on looking into the throat the tonsils may be seen red, prominent, and covered with mucus which is not easily detached. Sometimes the swelling is so considerable that the tumid glands fill up the space between the half-arches and leave but a small interval for the passage of food or drink. The lymphatic glands at the angle of the jaw are frequently swollen. Occasionally the inflammation extends from the tonsils to the salivary glands; the submaxillary and parotid glands swell, and ptyalism takes place. There is not much likelihood of confounding this, a form of *secondary parotitis*, with mumps, in which an outward swelling, visible beneath the ear, is found, but not a swelling within the throat, and in which no real difficulty in swallowing occurs, except, perhaps, when the tumefaction is at its height, and then only for a short time.

Tonsillitis terminates by resolution or by the formation of pus. There are no positive means of ascertaining that the inflammation is going to end in suppuration, although we may suspect that this will be the case when much pain is felt at the angles of the jaws and shooting to the ear, and when the symptoms have been severe and persistent for more than four or five days. Sometimes the pus may be seen through the covering of the tonsils; but often the vast sense of relief experienced by the patient, and the sudden improvement in deglutition, attended, perhaps, with an unpleasant taste, are the only signs that the collection of pus has been discharged. Attacks of tonsillitis are prone to be repeated, and may lead to permanent enlargement and induration of the tonsils. The enlarged tonsils, attended as they frequently are with cervical glandular swellings, may be mistaken for *cancer of the tonsils*. But in this affection sanious offensive discharges from the mouth occur, and, whether the disease be epithelioma or round-cell sarcoma, it extends rapidly; the neighboring lymphatic glands are early involved, the palate and the pharynx become impli-

cated, and hemorrhage is frequent, as are also difficulty of deglutition and attacks of suffocation.[1] Acute tonsillitis may be one of the manifestations of the rheumatic poison, and become associated with endocarditis;[2] it is also seen in connection with malaria.[3] At times the tonsils become gangrenous.[4] The primary lesion of syphilis may appear on the tonsils and present the ordinary sign of chancre,—ulceration with induration of surrounding parts, and enlargement of neighboring lymphatic glands, all yielding rapidly to antisyphilitic treatment.

Acute Follicular Tonsillitis.—There is a form of acute tonsillitis that is limited to the follicles and has well-marked clinical features. It begins with chilly sensations, to which a moderate fever, rarely exceeding 103°, succeeds. After a few days the fever disappears, a slight evening rise remaining, and in a week from the onset the patient is quite convalescent, though weak. At the height of the malady a swelling of the cervical lymphatic glands is often observed. But the characteristic feature of the disease is in the tonsils. These are red and slightly tumefied, and a thin yellowish or whitish punctiform exudation is seen in the crypts and around the follicular openings. This comes away gradually; in some parts much sooner than in others, and for days after convalescence from the general symptoms, the appearance is found in some follicles. When cast off, superficial ulcerations on the glands may be noticed. This form of tonsillitis is infectious, and various micro-organisms have been found in the exudate, particularly streptococci, staphylococci, and pyogenes aureus; also the pseudo-diphtheria bacillus.

Diphtheria.—There is another affection of the fauces which, in accordance with the clinical classification followed in this work, may be considered here, notwithstanding its specific character,—membranous angina, or diphtheria. Recent research leads us, indeed, to believe that the malady is primarily a local one, dependent upon the lodgement and multiplication of a specific bacterium. The constitutional symptoms are to be attributed to the absorption and action of the toxic products generated at the site of infection.

The bacillus of diphtheria was discovered by Klebs, and more fully studied by Loeffler. It is about as long as the tubercle bacillus, but nearly twice as thick. It is, as a rule, curved, but its form is variable.

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[1] Poland, Brit. and For. Med.-Chir. Rev., April, 1872; Newman, Amer. Journ. Med. Sci., May, 1892.

[2] See cases reported by Frederick A. Packard, Transactions of Association of American Physicians, 1899.

[3] Chassaignac, New Orleans Medical and Surgical Journal, Oct. 1888.

[4] Cragin, New York Medical Journal, Sept. 1888.

It has rounded extremities, which are sometimes club-shaped. It is non-motile; it does not form spores. It may be stained in cover-glass preparations with Loeffler's alkaline methylene-blue solution, which consists of thirty cubic centimetres of a concentrated alcoholic solution of methylene-blue and one hundred cubic centimetres of a 1 : 10,000 solution of potassium hydroxide. The organism grows best upon a culture-medium consisting of three parts of blood-serum and one part of a mixture of meat-infusion with one per cent. each of peptone and grape-sugar and one-half per cent. of sodium chloride. This is sterilized and at the same time solidified in test-tubes supported at an acute angle in a steam or hot-air sterilizer at a temperature a little below 100° C. The inoculation is made by means of a pledget of cotton wrapped on the end of a steel rod, and the culture-tube is kept in a

FIG. 52.

Klebs-Loeffler bacilli, from specimens prepared by Dr. Coplin and Dr. Bevan.

Fig. I. Fresh culture upon glycerin agar-agar. Eye-piece IV., Beck; Objective $\frac{1}{12}$ ol. im. Leitz.
Fig. II. Fresh culture upon blood-serum. Eye-piece IV., Beck; Objective $\frac{1}{12}$ ol. im. Leitz. This is also the appearance when obtained directly from the throat and subjected to the same power.
Fig. III. Old culture upon blood-serum. Eye-piece IV., Beck; Objective $\frac{1}{12}$ ol. im. Leitz.

thermostat at a temperature of about 37° C. In the course of from twelve to twenty-four hours dense, white, opaque colonies develop, and examination of cover-glass preparations will disclose the presence of the characteristic bacillus.

Bacilli resembling true diphtheria bacilli in appearance and in culture, but wanting in virulence, have been described as pseudo-diphtheria bacilli; but it is believed that these are merely a modified form of diphtheria bacilli, which, as Pasteur demonstrated, are variable in their infectivity.

The disease begins usually as sore throat, with redness and swelling of the arches of the palate, and of the tonsils. There is slight stiffness of the neck, the cervical and submaxillary glands of the jaw are enlarged and tender, and the subcutaneous tissues may become involved in the swelling. Within a period varying from a few hours to a few days, an exudation takes place on the tonsils, the uvula, and the soft palate. This exudation is more or less extensive, generally

tough, and of a white or grayish hue. It may show but little tendency to spread; or it may extend to the gums, and along the walls of the pharynx into the windpipe, sometimes even into the bronchial tubes and the lung structure. In some cases it passes upward into the nares, yet it may begin there or in the larynx simultaneously with its appearance in the throat. It usually appears, at an early stage, as a thin pellicle on the soft palate, and the uvula is apt to be œdematous.

The false membrane in the throat, once formed, darkens, wastes from the circumference towards the centre, and gradually disappears. But sometimes the coat becomes for a time thicker and thicker by the constant addition of fresh layers. This happens particularly in the "croupous form" of diphtheria, in which the inflammation is more intense from the onset and the fibrinous exudations succeed one another rapidly until the dense, thick coating of false membrane results. Under any circumstances, when artificially removed, the pseudo-membrane is soon redeveloped. After the first week from its beginning, no further exudation is apt to take place, and the danger arising from the membrane may be generally looked upon as over, unless, as is not uncommon, a relapse of the malady occur. The specific bacterium may, however, be present in the fauces, nasal passages, and maxillary sinuses for many days, even for weeks, after the disease has apparently come to an end.[1] It may be found occasionally when there is no false membrane, and also in the throats of healthy persons.[2] The Klebs-Loeffler bacillus, or the pseudo-diphtheritic bacillus, has been detected, too, in instances presenting all the features of follicular tonsillitis, and, however valuable a sign, it is still an open question whether the Klebs-Loeffler bacillus, in the absence of all clinical symptoms, can be accepted as an absolute test of the presence of diphtheria.

The constitutional symptoms vary greatly. The pulse may be frequent, the skin hot, and there may be much pain in the head; in fact, the symptoms are those of fever, with a temperature of 102° to 103°. Yet the temperature is exceedingly variable; there is often, even in the worst cases, an almost normal temperature. A sense of weakness and prostration are prominent from the onset. Not rarely

[1] Abel (Deutsche Medicinische Wochenschrift, 1894, No. 35, p. 692) has recorded a case in which virulent diphtheria bacilli were found sixty-five days after the onset of the primary illness; also Pearce (Journ. Boston Soc. Med. Sci., March, 1899).

[2] Feer, Correspondenz-Blatt für Schweitzer Aerzte, 1893, No. 8, p. 295; Welch, Amer. Journ. Med. Sci., Oct. 1894, p. 427 *et seq.*; Park and Beebe, Medical Record, vol. xlvi., No. 1247, p. 1.

the urine contains albumin and casts, and there may be partial or complete suppression of the renal secretion. In some instances typhoid phenomena manifest themselves, especially when decomposition of the disintegrating exudation takes place, giving rise to the septic form of the malady; in this the temperature may be even below the normal. The nervous system becomes much affected, and the tendon reflexes are lost.[1] In children exacerbations of pre-existing nervous symptoms may take place and give rise to a state resembling acute bulbar palsy.[2]

In diphtheria the danger is twofold: it arises partly from the depressing effect of the poison, increased as this effect must be by the absorption of toxic matter from the throat, partly from the mechanical obstruction caused by extension of the disease to the larynx and lungs. Again, at the height, or even at the decline of the malady, there is risk of heart-palsy or heart-clot, and of peripheral embolism.[3] Nor is the termination of the acute disorder always the termination of the complaint. A chronic irritation of the throat, lasting weeks or months, and readily relapsing, on exposure to infection, into a diphtheritic sore throat, remains; or albuminuria, which outlasts the acute manifestations; or pleurisy, or bronchitis and pneumonia,—both of which may be delayed until after the exudation has disappeared from the throat,—increase the list of the complications of the affection, and protract or imperil the convalescence. Occasionally, too, inflammation of the joints is observed in the course of diphtheria, or as a sequel, and sometimes trophic changes in the periarticular structures are met with.[4]

Some morbid conditions there are which may be wholly looked upon as after-symptoms. A paralysis of the velum palati and of the pharyngeal arches, making itself apparent by a peculiar nasal intonation of the voice, and by proneness to regurgitation of fluids through the nostrils, is among the earliest of them; it manifests itself often, indeed, just at the termination of the acute malady. Later appear impairment of vision, gastrodynia, ulcers in various parts of the body, profound anæmia, and that gradual failing of muscular power with anæsthesia, and absence of reflexes, that bespeaks diphtheritic paral-

[1] McDonnell, Medical News, Oct. 15, 1887.

[2] Guthrie, Lancet, April 18, 25, 1891.

[3] A case has been recorded in which embolic obstruction of the popliteal artery occurred during convalescence from an attack of diphtheria, and amputation of the affected member became necessary. Rooney, Occidental Medical Times, vol. vii., No. 4, p. 188.

[4] Lyonnet, Lyon Medical, Jan. 4, 11, 1891.

ysis. In rare instances these symptoms occur early in the attack.[1] They are attributable to the development of a peripheral neuritis dependent upon the action of the toxic products of the disease. Hemiplegia has been observed in some cases as a result of rupture of a cerebral blood-vessel or its occlusion by a clot.[2] Other symptoms of profound nervous derangement have also been recorded, such as peripheral neuritis in which the sense of smell and the muscular sense were lost and profound impressions were referred to corresponding points on the opposite side of the body,[3] or with temporary absolute deafness, unsteadiness of gait, and paralysis of the palate.[4] Furthermore, I have known aphasia to follow the depressing complaint.

But to look at the differential diagnosis of the disorder. It varies widely from stomatitis, from tonsillitis, from pharyngitis,—in truth, from all the ordinary local inflammations of these structures,—by the presence of a membrane, by the striking constitutional symptoms, and by the sequelæ. The diagnosis becomes unequivocal if, in addition to these, the characteristic bacilli are found on bacteriologic examination of some of the material taken from the throat or the nose.

Yet there are certain sources of error against which it is necessary to guard. In *simple pharyngitis*, a mass of mucus, in part derived from the nares, is apt to collect on the inflamed membrane, and looks at first sight like the coating from an exudation; but it may be easily removed, and a closer inspection proves its true nature. In *follicular tonsillitis*, liquid may ooze from the openings of the follicles on the surface of the swollen tonsils, or little yellowish or whitish points form there. But they are strictly confined to the gland, exhibit no tendency to spread or to coalesce, and are generally small white specks of roundish or oval shape. These appearances constantly occasion mistakes, especially as regards mild cases of diphtheria. What adds to the difficulty is that follicular tonsillitis is contagious. Should, in an individual instance, the facts mentioned be insufficient to solve the doubt, the microscope can do so; for it shows the white or yellowish masses to be largely composed of epithelium, with streptococci and staphylococci in abundance, but not with the true Klebs-Loeffler bacillus.

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[1] As in two cases reported by Dabney, Medical News, Jan. 16, 1892, in which they appeared on the first and second days respectively.

[2] McPhedran, Canadian Practitioner, 1892, No. 19, p. 454; Allen A. Jones, Medical News, Oct. 22, 1892, p. 467; Edgren, Deutsche Medicinische Wochenschrift, 1893, No. 36, p. 864; C. W. Sharples, Medical News, Aug. 4, 1894, p. 124.

[3] Gay, Brain, part lxiii. p. 431.

[4] Tooth, British Medical Journal, 1893, No. 1680.

Ulcerative stomatitis, the form of stomatitis most likely to be confounded with diphtheria, and especially with this malady when the exudation lines the gums, is discriminated by the ulceration or sloughing; whereas the mucous membrane in the pseudo-membranous disease remains intact, save in the rarest instances. The same feature distinguishes diphtheria from *gangrene of the mouth*, for which, on account of the extreme fetor of the breath, it is sometimes mistaken, and aids in distinguishing it also from other kinds of stomatitis, as from *thrush*. In the latter, too, the buccal mucous membrane, and not the throat, is chiefly affected, and the abdominal symptoms, and the other constitutional phenomena, are different. So are they in *aphthæ*, in which, moreover, the superficial ulcerations, which bleed when touched, the unbroken vesicles or pustules in other parts, and the seat of the disorder—usually on the edge of the tongue, on the internal surface of the lips, and on the gums and inside of the cheek—are points to be taken into account.

Besides these affections, there are others which must be distinguished from diphtheria. We occasionally find cases occurring in epidemics, and where the membrane is limited nearly altogether to the follicles, and chiefly to the tonsils. As the membrane passes away, ulcerations are obvious. Swelling of the glands of the neck, and fever, but not of acute type, attend this *ulcero-membranous angina*, which, moreover, shows a strong disposition to relapses. But, though kindred to diphtheria, and in isolated instances perhaps difficult to discriminate, it differs from it in its seat and in its want of tendency to spread, in the formation of superficial ulcers, in its less marked constitutional depression, and in its invariably favorable termination.[1] It is similar to herpes of the tonsils, described by Trousseau. In acute inflammation of the fauces it is not unusual, especially in certain families, to find a form of exudation on the surface of the throat due to excessive desquamation of the superficial layer of the epithelium of the inflamed mucous membrane. But a light rubbing with a cotton tampon removes it, and shows a surface of mucous membrane which is not bleeding or ulcerated. The false membrane of diphtheria is so adherent to the subjacent tissue that it cannot be wiped off, and, if removed forcibly, will leave a bleeding surface and soon be reproduced. Then, as already stated, there are cases of membranous, or ulcerated, sore throat with membranes that are not diphtheritic, in which the Loeffler bacillus is absent, and various forms of strepto-

[1] See a paper in the Amer. Journ. Med. Sci., July, 1870, in which I have described an epidemic of the kind.

cocci, staphylococci, and pseudo-diphtheritic bacilli are found in the membrane, as in instances observed among soldiers by Cassedebat.[1] Energetic treatment should be, however, promptly instituted, since these common false membranes may insidiously prepare the way for the culture of the diphtheria microbe. They also, by entering the crypts of the tonsils, may lead to the frequent recurrence of this form of sore throat. In the appearance of the false membrane there is nothing clinically distinctive. Whether there be not still other kinds of membranous sore throat to be separated from true diphtheria is a matter requiring investigation. The pseudo-membranous inflammations of the throat attending scarlatina and measles and other of the exanthemata have been shown not to be diphtheritic, although they seem to predispose to invasion by the diphtheria bacillus.[2]

There is an acute disease of the throat to which Todd especially has called attention,[3] and which presents also some strong points of similitude to diphtheria,—*erysipelas of the fauces*. Like diphtheria, it is a most dangerous ailment; as in diphtheria, the morbid process may extend to the larynx, the mucous membrane be swollen and exhibit a peculiar dusky-red color, the poison paralyze the muscles of the palate and pharynx, and liquids be rejected through the nostrils and mouth. But the difficulty in deglutition differs from that of diphtheria in being present from the onset, and is not attended with enlargement of the glands of the neck, or with the formation of a false membrane. If the erysipelatous inflammation extend to the larynx, there is local pain, with urgent dyspnœa and hoarseness, and usually rapid exhaustion supervenes. In cases of the kind, the submucous tissues of the larynx are found extensively infiltrated with pus. Erysipelas of the fauces may happen without erysipelas showing itself on any external part of the body; on the other hand, erysipelas beginning in the fauces may spread to the face.[4]

This erysipelas of the fauces is not a frequent disease; and it must be stated that there are cases of diphtheria which simulate it very closely. I have seen a number of instances of the malady in which the whole mucous membrane was of a vivid or dusky hue; in which there was much swelling, with an effusion of serum, especially in the submucous tissue of the uvula, causing it to look like a small trans-

[1] Des Angines Couenneuses non Diphthériques, Arch. Gén. de Méd., 1897, p. 385.

[2] Booker, Bulletin of the Johns Hopkins Hospital, vol. iii., No. 26, p. 129; Park and Beebe, Medical Record, vol. xlvi., No. 1247, p. 1.

[3] Clinical Lectures on Acute Diseases.

[4] Cases quoted in Schmidt's Jahrbücher, 1869, No. 1.

parent bag; in which immense difficulty or even impossibility in deglutition existed,—yet in which no membrane appeared for days after the violent inflammation of the throat had set in, and was, when it showed itself, very slight in extent, and out of all proportion to the inflammation. But the constitutional symptoms and the sequelæ were the same as those of ordinary diphtheria. In one of the cases of the kind referred to, suppuration of one of the tonsils took place in consequence of the inflammation; a layer of deposit had coated parts of the tonsils and of the half-arches and uvula.

How shall we separate diphtheria from *membranous croup?* In the great majority of instances there is no separation, for membranous croup is laryngeal diphtheria. But there may be a membranous croup that is not; such as follows scalding the throat, irritant poisons, violent laryngitis, or is seen at times in the exanthemata. Now, in cases of non-diphtheritic membranous croup, the disease affects almost always primarily the windpipe. The reverse is the rule in laryngeal diphtheria: it extends from the throat. Further, ordinary membranous croup is not contagious, as diphtheria is. The finding of the specific bacillus in the false membrane in a doubtful case establishes the diagnosis.

On one symptom we cannot lay as much stress as might be supposed: Albuminuria, the elaborate report of the committee of the Medico-Chirurgical Society has taught us,[1] is not always present in laryngeal diphtheria, owing to the early fatality of the malady; again, in certain cases the mere dyspnœa of laryngitis may give rise to albumin in the urine. Yet when albuminuria is marked, and when it has happened where an affection of the fauces has preceded the laryngeal implication, it points to an infective cause,—to laryngeal diphtheria.

Lastly, diphtheria may be confounded with *scarlatina.* When, indeed, we reflect on the similar appearance of the throat, on the occurrence of albuminuria in both maladies, and on the frequency with which both are found to prevail at the same time as epidemics in a community, it is not astonishing that one should be looked upon as but a modified form of the other. Allied they certainly are, but not identical; for the poison of one leads to a thoroughly defined rash, and leaves a protective influence against a second attack, and often also deafness, suppuration of the glands of the neck, and dropsy,—phenomena which are not encountered in the other. It is true that in very rare instances of diphtheria we encounter a slight erythema

[1] Medico-Chirurgical Transactions, vol. lxii., 1879. Some of the anatomical points involved are also well discussed by Weigert in Virchow's Archiv, vols. lxx. and lxxi.

of the neck and breast, but it is not like the vivid, diffused rash of scarlet fever. Moreover the exudation in the throat is not exactly similar in the two diseases. In scarlatina it is pultaceous, and not coherent, and has no tendency to spread to the respiratory passages. Bacteriologic examination, further, may disclose the presence of streptococci and staphylococci, but not the bacillus peculiar to diphtheria. Then the albuminuria happens at a different period. In scarlatina it is a sequel rather than a concomitant; in diphtheria it is a concomitant rather than a sequel. Further, the gravity of the symptom is not the same. In the latter malady it is an indication of danger; it has not so serious a meaning in the former.

Diphtheria may be intercurrent in various maladies: in typhoid fever, in the exanthemata, in pneumonia. A microscopic examination and culture experiments can alone settle whether the membranes are truly diphtheritic or only formations of false membranes. The exudation in diphtheria is not always restricted to the throat. It may show itself in a wound or on excoriated skin, on the nasal mucous membrane, the conjunctiva, the nipple, the uvula, or around the anus; it may be found coating the stomach, the intestines, and the ramifications of the bronchial tubes.

Nasal diphtheria is a very grave form of the malady: it may either be present alone, or coexist with a deposit in the fauces and pharynx. It generally occurs with evidences of the septic form; the symptoms are of a low type, and we recognize the affection by carefully inspecting the posterior pharynx and seeing that the membrane extends upward; by noting the irritated, reddened look of the nostril, even when no membrane can be discerned in it; and by the coryza, the sense of obstruction in the nose, and the acrid sanious discharge which comes from it. In cases in which the nasal duct and the lachrymal canal are stopped up by the false membrane, tears are constantly rolling down the cheeks. Epistaxis is a not uncommon symptom; swelling of the cervical glands may or may not be present. Recent bacteriologic investigation has shown that so-called membranous rhinitis is in reality often of diphtheritic origin.[1] And in the enlarged glands in any form of diphtheria the characteristic bacilli are found in the opaque, yellowish masses, consisting principally of fibrin, which they contain.[2]

Mumps.—This, like diphtheria, is a general disease, and is only here described as a matter of clinical convenience. Parotitis is most

[1] Abbott, Medical News, May 13, 1893, p. 505.

[2] Bulloch und Schmorl, Beitr. zur Pathol. Anatomie, etc., von Ziegler, B. xvi. H. 2; Centralblatt für Innere Medicin, 1895, No. 6, p. 156.

commonly seen as an epidemic malady; but we occasionally encounter a secondary parotitis following typhus fever, scarlet fever, smallpox, measles, and dysentery. In this form suppuration is much more common than in ordinary mumps. The disease generally begins with pains at the angle of the jaw, which are soon followed by a marked swelling, first on one side, then on the other, that results in the head being kept rigid. The tumid glands are sore, and become more painful during attempts at swallowing and chewing, though there is really little, if any difficulty in swallowing. If the patient be made to swallow slowly ten to thirty drops of undiluted vinegar, decided pain is produced in the affected glands,—an old and useful diagnostic test, to which Dr. Louis Starr called my attention. The mouth is generally filled with saliva, though it may be very dry; and the hearing may be impaired, or, for the time being, entirely lost, and ringing in the ears is very common. The temperature generally ranges between 101° and 102°, but in cases of orchitis following mumps, or of metastasis, I have seen it 104° to 105°. The nervous system may become decidedly affected, and the action of the heart weak and irregular. Acute mania has been known to become associated with mumps; so has peripheral neuritis.[1] Parotitis is easily recognized. There is no swelling of the tonsils, hence it cannot readily be mistaken for tonsillitis. Laveran and Catrin have found a diplococcus in mumps, in the secretions of the parotid and other glands, as well as in the blood.[2]

In cellulitis of the neck, *angina Ludovici*, the swelling may mislead, but it is uniform and not confined to the region of the parotids; the constitutional symptoms are very severe, pointing to an infective malady. Ludwig's angina is met with as an idiopathic affection, or in certain fevers, such as scarlet fever or diphtheria.

Chronic Sore Throat.—Attacks of angina are prone to recur, and to lead to chronic inflammation of the structures. Now, an affection of this kind is liable, on any exposure, to be kindled into the acute complaint; besides, it yields at all times some manifestations of a disorder of the throat. A thickening of the folds of membrane forming the half-arches, a tumefaction of the follicles at the upper part of the pharynx, a lengthening of the uvula, are the visible signs of the chronic malady; a constant disposition to clear the throat, and a dry cough, are often the attending general symptoms. Owing to the habitual coughing, the patient may be suspected to be laboring under phthisis, and be treated accordingly, when the whole difficulty lies not in the lungs, but in the throat. Yet an error in the opposite direction is per-

[1] Lancet, April 9, 1887.

[2] Gazette Médicale, June, 1893.

haps more frequently committed. Tonsils and uvulas are removed, with the view of curing a cough which is really kept up by a source of disturbance in the lungs, in forgetfulness of the fact that, in scrofula, and tuberculosis, chronic enlargement of the tonsils and follicular pharyngitis are by no means unusual. A careful examination of the chest and a bacteriological examination of the sputum ought always to be made, even when inspection of the throat shows disease to be there present.

The follicular disease of the throat, or "clergyman's sore throat," is the most frequent of all the morbid conditions which produce a chronic sore throat. The abnormal condition of the follicles of the pharynx and fauces often extends to the larynx. There are constant hawking and attempts at clearing the throat, and not infrequently roughness of voice or decided hoarseness. On inspecting the throat, the enlarged mucous follicles can be readily discerned; those on the pharynx are very prominent. In cases of long standing, the follicles may ulcerate, and very commonly they pour out an acrid secretion. But, unless from coexisting enlargement of the uvula, or an altered position of the epiglottis, or a laryngeal or bronchial complication, there is no decided cough. The follicular disease may occur in consequence of repeated attacks of sore throat, or be an attendant upon gastric disorder, or follow constant over-exercise and straining of the voice.

Chronic rheumatic sore throat gives rise to pain which is often referred to the hyoid bone, is increased by pressure, and is also felt in the tonsils. Ingals[1] points out that the pain often entirely disappears while the patient is eating, but increases in cloudy and damp weather. There are signs of slight congestion in the throat, and generally in the larynx, yet mostly out of all proportion to the pain. The general health remains good, and we find no fever; there is apt to be a history of a rheumatic diathesis.

Ulcers are not often developed in the fauces during an attack of acute inflammation, except in the specific sore throat of scarlatina; in chronic inflammation, especially if occurring in scrofulous persons, they are more common. The most profound ulcerations are those of constitutional syphilis, implicating, as they do, not only the tissues of the fauces, but also the parts in front, and destroying both the fleshy covering of the bones and the bones themselves. With regard to treatment and to prognosis, it is of the utmost importance to distinguish these *syphilitic ulcers* from those produced by other causes. The coexistence of a cutaneous eruption of a syphilitic character, and

[1] Medical News, March, 1890.

enlarged lymphatic glands, or the history of antecedent syphilis, would lead us to a correct conclusion; but an accurate history of a syphilitic infection cannot be always obtained. The ulcers are not superficial and stationary, like those resulting from ordinary inflammation, but are deep and have a strong tendency to spread. They are rounded, or of a serpiginous form, with borders well defined and elevated; and the inflammation which precedes them is limited to spots, and is not so diffused, nor attended with so much swelling, as the inflammation that exists prior to simple ulceration. The primary lesion is occasionally met with, cases of *chancre of the tonsil* being well known to syphilographers. Syphilitic ulcers must be distinguished from the deep ulceration with spreading destruction of tissue that occurs in *cancer of the tonsils*.[1]

PHARYNX AND ŒSOPHAGUS.

In describing the affections of the fauces, those of that portion of the pharynx which is most usually the seat of disease have been at the same time described. Indeed, when we speak of acute or chronic pharyngitis, we generally mean acute or chronic inflammation of the fauces, to which the upper part of the pharynx belongs. Inflammation of the portion of the pharynx which is out of sight when the tongue is depressed is rare. It may be presumed to exist if there be pain and an impediment in swallowing when the food arrives opposite the top of the larynx, while the respiration remains free and the voice unaffected. Abscesses sometimes form between the textures composing the pharynx, and between its posterior wall and the cervical vertebræ. These *retropharyngeal abscesses* mostly result from disease of the vertebræ. They occasion great difficulty in deglutition and in breathing; an altered voice; dull pain and stiffness in the neck; external swelling, which may or may not be œdematous; and commonly a tumefaction at the back of the throat, which can be seen, or which can be felt with the finger pressed against the posterior wall of the pharynx. On account of the obstructed respiration and the changed voice, the disease is liable to be mistaken for croup. Its differences have been already enumerated. Retropharyngeal abscess is often confounded with coryza and tonsillitis. It differs chiefly from tuberculosis of the retropharyngeal glands by the presence of tuberculous lesions of the deep lymphatic glands of the neck.[2] It may happen in infancy.[3]

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[1] See Newman, Amer. Journ. Med. Sci., May, 1892.

[2] Sokoloff, Vratch, May, 1891.

[3] See cases of Pollard, Lancet, Feb. 1892.

There is a peculiar form of pharyngeal disease due to the accumulation on the mucous membrane of a micro-organism generally supposed to be the leptothrix, though Hemenway[1] in his elaborate article pronounces it to be the bacillus fasciculatus. The deposits in this *pharyngo-mycosis* take place largely in the follicles.

Œsophagus.—The œsophagus is not often the seat of disease. We meet with acute inflammation produced by swallowing boiling water or corrosive poisons, especially nitric or sulphuric acid, or ammonia. The symptoms of acute *œsophagitis* are usually mixed up with those of inflammation of the pharynx or of the stomach. We may, however, infer its presence if difficulty and pain in deglutition exist for which nothing in the throat accounts, and if these phenomena be associated with hiccough and with a burning sensation between the shoulders, in the course of the tube. Œsophagitis is sometimes encountered in infancy.

Of the chronic diseases of the œsophagus, *stricture* is the most common. The narrowing may take place at any part of the tube, and results from preceding inflammation or ulceration, from cancerous degeneration of the walls, from polypoid growths projecting from the mucous membrane, or from the pressure of a tumor, of an abscess, or of an aneurism; sometimes it is congenital. The formidable malady manifests itself by an impediment in swallowing; even liquid food cannot pass without great difficulty; and if the stricture go on increasing, the patient perishes miserably by starvation. In addition to the obstruction to the passage of food, we may find a peculiar pain occurring at a particular part of the tube, and the patient raises, without cough or vomiting, clots of blood presenting the shape of the stricture.

The matter ejected in the attempts at deglutition consists simply of masticated food together with more or less mucus, and, unlike what comes from the stomach, has an alkaline reaction. If long retained, the albuminous materials are macerated; the starchy materials are in process of fermentation; fungi are formed in great quantities, although never sarcinæ.[2] By applying the stethoscope posteriorly, between the shoulders and at the lower part of the neck, while the patient swallows a mouthful of water, a peculiar sound is heard when the water passes through the narrowed portion of the tube. Should there be doubt as to the seat of the obstruction, a bougie will clear up the doubt; and thus we possess in this instrument the most valuable diagnostic as well as therapeutic agent. But we must not immediately conclude, because

[1] Journal of Laryngology, Feb. 1892.

[2] Ziemssen, "Diseases of the Œsophagus," in Ziemssen's Cyclopædia.

the bougie meets with resistance, that an organic stricture is present. The narrowing may be only *spasmodic*, yet give rise to the symptoms of organic constriction. But they are not permanent: at times nourishment is readily swallowed, and a full-sized bougie passes with ease. Spasmodic stricture occasionally accompanies ulceration of the larynx: but it is chiefly met with in hypochondriacs and in hysterical women. The latter, indeed, sometimes fancy that they are incapable of swallowing, and reject the food they take without there being even a temporary spasm to prevent its passage. Spasmodic stricture is also observed in hydrophobia and as an attendant on cerebral disease.

The distinction of the other causes of stricture is not always an easy matter. In the stenosis arising from *syphilis*, we lay great stress on the history. In the strictures due to *compression*, we discern the swelling that has occasioned them, and the œsophagus is apt to be pushed to one side. In strictures the result of *cicatrices*, we have the gradual development of the affection after an injury or the swallowing of some irritant poison, and the great resistance of the dense tissues to the sound is very significant. *Cancerous narrowing* occurs after forty years of age, progresses steadily, and, as Ziemssen has pointed out, is frequently associated with paralysis of the recurrent laryngeal nerves. It may affect the whole middle part of the œsophagus.[1] Cancer of the œsophagus is most commonly epithelioma. We may get great aid in the study of these organic diseases of the œsophagus from the X-rays. They will also show us readily whether a foreign body is present, or whether the signs of obstruction are due to the pressure of an aneurism.

Rupture of the œsophagus may be met with as the result of protracted vomiting or the introduction of bougies. The accident is apt to occasion great pain. It leads to a rapidly fatal result.[2]

Dilatation of the œsophagus above the seat of a stricture, or without a stricture existing, is, on the whole, a rare disease. Its chief symptoms when extensive, are difficulty in swallowing, vomiting, or regurgitation of food, a swelling in the neck coming on after eating and diminishing greatly after vomiting or by pressure, slowly progressing inanition, and at times long spells of delusive improvement. The sound may penetrate through the neck of the sac with difficulty, or enter it readily, which largely depends upon whether the sac be empty or full; once in the sac, the end of the tube can be generally moved about with ease.

[1] Moore, Lancet, London, 1883, i. 13.

[2] See for cases, paper by Fitz, Amer. Journ. Med. Sci., Jan. 1877.

In all the diseases mentioned, the value of the *sound* as a means of diagnosis has been spoken of. A few more remarks about it may not be amiss. Great care should be always used in passing a sound. The patient's head should be well thrown back, and the instrument passed along the posterior wall of the pharynx with the utmost gentleness. There is a slight resistance as it goes past the cricoid cartilage. When an aneurism or an organic disease of the heart exists, it should not be employed at all. When the sound on reaching a particular spot always occasions pain, we may infer the existence of inflammation or ulceration at this point, and, in the case of ulceration, some pus or blood is likely to be brought up on the instrument. Should any doubt exist whether the sound has passed into the œsophagus or into the larynx, let the patient be directed to speak; he can make no noise if the tube be in the larynx. In cases remaining doubtful, a lighted candle may be placed before the end of the tube projecting from the mouth. If the instrument be in the windpipe, the flame will be wafted to and fro with the currents of air; if in the œsophagus, this is not observed, except when the tube is in the intrathoracic portion.

The diseases of the œsophagus may be studied by means of *auscultation*, listening while the patient swallows food or liquid; and we owe to Hamburger an elaborate description of the sounds.[1] In health, the œsophageal sound is extremely distinct, but of very short duration; the pharyngeal swallowing sound is generally a loud gurgle. In a moderately advanced stage of stricture of the œsophagus, a noise similar to emptying a bottle, "clucking," "gurgling," is perceived; while in cases of dilatation we are apt to meet with a sound like that heard when rain driven by the wind impinges on a solid and is deflected. In cases of very marked stricture or of obstruction by an impacted foreign body, we find that the act of deglutition cannot be detected below a certain point, while it is distinct above. To auscult the œsophagus, the stethoscope should be placed in the vicinity of the hyoid bone, also to the left of the vertebral column from the upper dorsal vertebra downward. This method of exploration has not, however, proved itself of much value. Some cases show that the phonendoscope is of greater service.

The disorders of the pharynx and œsophagus have as a common symptom difficulty in swallowing. But we must not forget that other causes may produce *dysphagia*, such as paralysis of the muscles of the throat, diseases of the larynx or trachea, particularly ulcerative diseases, and aneurismal tumors within the chest.

[1] Jahrbücher der k. k. Gesellschaft der Aerzte in Wien, Bd. xviii.

CHAPTER VI.

DISEASES OF THE ABDOMEN.

The abdominal cavity contains viscera of very varied functions: some form, others break down organic constituents; while others, again, excrete the broken-down material. They all, however, labor in one cause; they all work towards preserving a normal state of the blood, either by preparing fit matter for it or by removing such substances as would be hurtful if they were retained. Any serious derangement of any of these viscera, especially any serious chronic derangement of those which are not simply reservoirs, must therefore lead to a deterioration of the blood and to a defective nourishment of the body. But these symptoms furnish but little information as to the particular organ at fault. This we learn to some extent by examining, where it can be done, the secretions or excretions; to some extent by noticing the peculiar appearances of the skin which are produced by alteration of the blood; and by the exploration of the organs through the parietes of the abdomen. It is, in truth, by means of the physical method of investigation that we often obtain the most valuable information, not only as to the seat but even as to the nature of the morbid action; and, although physical exploration of the abdomen does not yield as perfect results as when applied to the affections of the thorax, it still supplies us with an amount of knowledge most valuable, and with which it would be difficult to dispense. Let us pass in review the different methods of physical diagnosis with reference to abdominal disorders.

Methods and General Results of Physical Examination of the Abdomen.

INSPECTION.

By inspection we learn the size, shape, form, and movements of the abdomen. To inspect the abdomen satisfactorily, the patient should be placed in an easy attitude, preferably either standing or sitting. Whenever practicable, ocular inspection must be made not only from the front, but also from the sides and from the back. In

appreciating the results thus obtained, it is necessary to bear in mind that the appearance of the abdominal walls is modified by certain physiological conditions. The abdomen is much larger, in comparison to the size of the chest, in childhood than in adult age. It is more voluminous in women, especially such as have given birth to children. It increases in size with advancing years, particularly when a tendency to obesity exists. Its shape is somewhat altered by the pernicious habit of wearing tight stays. Its upper portion is distended after a copious meal.

In disease we may observe either partial or general *abdominal enlargement*. The latter is caused by accumulations of air in the intestinal canal; by liquid in the peritoneum; by an œdematous or obese condition of the abdominal walls; or by large tumors which fill up the whole cavity. A partial enlargement is mainly produced by an increase in size of particular organs, or by swelling of the mesenteric glands, or by tumor,—solid or hernial; and it is sometimes due to diseases above the diaphragm. A pleuritic or a pericardial effusion, or emphysema of the lungs, may give rise to a marked fulness below the margin of the ribs. The condition known as *enteroptosis*, or *splanchnoptosis*, in which there is undue freedom of movement of the abdominal viscera, may reveal itself to inspection by the flaccidity and thinness of the abdominal parietes and their protrusion in the upright posture. Sometimes, also, the outlines of the viscera may be distinguishable.

A *retraction of the abdominal parietes* is perceived in general emaciation, and is very obvious in that dependent upon a narrowing of the cardiac or the pyloric orifice of the stomach, or upon chronic diarrhœa or dysentery. It is also noticed in lead colic and in cephalic diseases, especially in tubercular meningitis.

There are further changes in the appearance of certain external parts which tend to elucidate the state of the parts within. Distention of the superficial veins indicates that an obstruction to the flow of blood exists in the large veins of the abdomen, either in the portal system or in the vena cava. The lessening of the depression at the umbilicus, unless it be produced by pressure limited to the spot where the umbilicus lies, is a sign of general abdominal enlargement.

While inspecting the abdomen, we may see distinct *movements*. The act of breathing gives rise to motion which is very slight when a tumor or any other impediment interferes with the free action of the diaphragm, and which is much exaggerated by diseases within the thoracic cavity. The rolling of the intestines is sometimes visible on the exterior; so are at times those shiftings of accumulations of gas

which give rise to a series of jerking elevations; so, too, are occasionally the spasmodic contractions and relaxations of the abdominal muscles. But none of these is as frequently encountered as a pulsation in the epigastric region. The inspection of internal organs, such as the stomach, will be considered in connection with those organs.

PALPATION.

We judge by the application of the hand of the size, position, and consistence of the viscera which are felt through the abdominal walls. We determine whether the parts are firmly attached or movable; whether they are smooth or nodulated; whether they possess a motion of their own; whether they are tender; and by tapping with the fingers of one hand, while those of the other are applied to another portion of the surface, we discover, by the peculiar feeling of fluctuation, the presence of fluid in the abdominal cavity. We satisfy ourselves further, by the sense of touch, of the existence and outlines of abdominal tumors, and of the state of the parietes, whether resistant or elastic, œdematous or not; and we may detect a friction fremitus.

In order to use palpation with most effect, the abdominal muscles must be relaxed; and to do this the patient should be placed on his back, and the thighs be flexed on the body. Occasionally it is essential to vary this position; to turn him from side to side, or to examine him when erect. The amount of pressure, too, should not always be the same. When we wish to examine deep parts, the pressure is increased. The character and the intensity of the pain that pressure calls forth often throw considerable light on the disease we are investigating. Thus, if it take deep pressure to produce pain, we are usually right in concluding that the mischief is not superficially seated. The pain of inflammation of the serous membrane is commonly much augmented by pressure, and is of a severe, cutting character. Pain due to inflammation of the mucous membrane of the intestinal tract is duller. All neuralgic or nervous pain, such as that of colic, is, as a rule, relieved rather than increased by pressure, and may be thus distinguished from inflammatory tenderness. One or both hands may be used in the practice of palpation, and sometimes shock-like manipulations will reveal conditions not otherwise discoverable.

Palpation is used as a means of diagnosis by the introduction of the hand into the rectum. But the method is both disagreeable and not free from danger. Dilatation of the sphincter should be gradual, five minutes at least being allowed for its accomplishment. And, with all precautions, the information obtained may be indecisive. Strictures in the rectum or in the sigmoid flexure of the colon may be readily

discerned, but a stricture at the lower part of the descending colon may exist, though the hand be unable to discover it.

We might with palpation consider the results obtained by the use of bougies and of tubes, such as the stomach-tube. But these will be more appropriately considered when discussing the diseases of individual organs.

PERCUSSION.

Percussion is, in the study of abdominal affections, only less valuable than palpation. By it we can circumscribe the different organs with accuracy; we can judge of the position of the stomach and intestines; we can limit the distended bladder, and fix the borders of the liver and spleen. By its aid, further, we can tell whether a distention of the abdomen is produced by air, or by a solid tumor, or by liquid. But, without entering here into particulars as to its use in individual disorders, we shall examine the results when applied to the healthy abdomen.

To render percussion a trustworthy interpreter of the state of the abdominal viscera, the patient should be placed in the same position as for palpation. The sounds are best elicited by mediate percussion, and where great accuracy is desirable, we may advantageously make use of auscultatory percussion or the phonendoscope. But for correct deduction we must be acquainted with the relations of the parts which the abdominal walls conceal from view, and take into account that during the digestive process the contents and position of these organs may vary sufficiently to modify the percussion sound.

To begin with the airless viscera. The *liver* is one of the easiest organs to limit. We determine its upper boundary by striking with moderate force in a line from somewhat above the right nipple towards the lower part of the thorax, until marked resistance and dulness tell us that a solid organ has been reached. At this point we make a mark; then we percuss downward from near the median line, and above the dulness just obtained; then from the axilla downward; then posteriorly from beneath the lower angle of the scapula; and so on, until the line traced reaches the vertebral column.

The dulness thus elicited marks the upper boundary of the liver; at least of the portion more directly in contact with the abdominal walls. Anteriorly it extends from the lower extremity of the sternum to between the fifth and sixth ribs; at the side, the dulness is generally in the seventh intercostal space; near the vertebral column, it is on a level with the tenth or the eleventh, more rarely with the ninth, interspace. The dulness of the left lobe reaches nearly two inches across the median line; but the heart lies here so near to the liver that

we cannot with accuracy distinguish the flat sound of the one from the flat sound of the other.

After the upper border has been fairly traced out anteriorly, laterally, and, if thought necessary, posteriorly, we determine the inferior margin of the organ. This is readily effected by percussing downward from the already ascertained line of dulness, and noting where the large intestine sends forth its distinct tympanitic sound. To determine the lower border correctly, the pleximeter must be pressed firmly on the integuments, and the stroke of the finger be slight; for if it be strong, we obtain the sound of the surrounding hollow viscera through the thin layer of liver which covers them, and before we have arrived at its margin. This mode of procedure is different from the one pursued to determine the height to which the liver rises, because the position of the parts is different. Superiorly, the lung descends between the surface and that portion of the convex surface of the liver which fits into the diaphragm, and it requires strong percussion to bring out the dulness of the deep-seated solid organ. By forcible percussion we detect a decided loss of the pulmonary resonance at about the fourth intercostal space.

The inferior border of the liver, anteriorly, is generally found to lie immediately at, or to project below, the last rib; posteriorly, we cannot determine this border positively, for it becomes continuous with the dulness occasioned by the right kidney. The lower margin of the left lobe is commonly met with at the upper third of a line drawn from the ensiform cartilage to the umbilicus. A distended gall-bladder may cause a strictly outlined dulness below the surrounding liver. The percussion dulness of the liver is everywhere lowered by a full inspiration.

The *spleen* is not so easily circumscribed as the liver. Indeed, if the stomach or the intestines be distended, it is difficult to detect the dull sound of the spleen. To find its limits, we must place the patient on his right side, with his legs flexed; or let him stand erect, and then begin to strike with some force in a line from the axilla to the crest of the ilium. At the ninth, or sometimes at the tenth, rib, the sound becomes dull, and there is much greater resistance to the finger. Here is the upper boundary of the spleen. We mark the spot, and continue to percuss in the same line until, at about the twelfth rib, we arrive at the lower boundary of the organ, as indicated by the distinct tympanitic sound of the intestines.

After the vertical diameter has been thus ascertained, the horizontal is readily determined by percussing from the median line to a point between the lines which trace the superior and inferior margins, and

by noticing where the sound of the stomach gives way to the dull sound of the solid viscus. When these three points have been decided upon, we have learned enough for practical purposes. We may then, if we choose, percuss posteriorly; but we cannot circumscribe the spleen with any accuracy behind, because its dulness becomes continuous with that of the left kidney.

The average size of the spleen is four inches in length and three in width; but it may in a diseased state increase to twice or three times that size. When the viscus eludes detection by percussion, we may infer it to be small; provided the stomach and intestines be not much distended with gas.

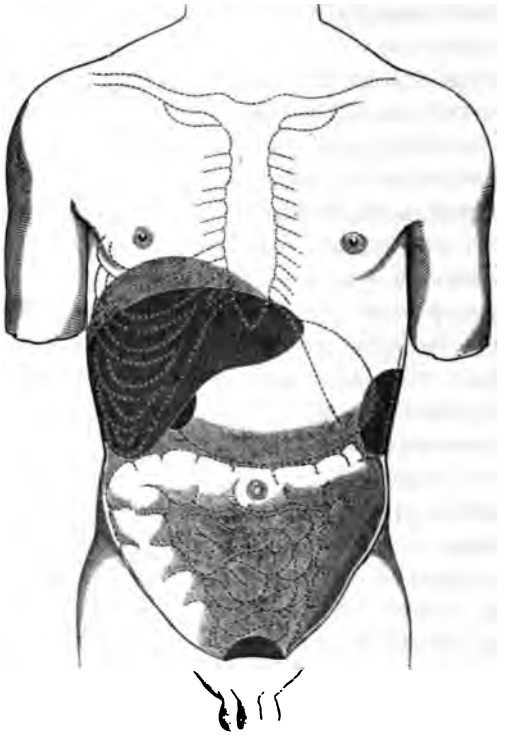
The *kidneys* cannot be limited with anything like accuracy, except at their inferior and outer borders, where the dull sound they occasion is surrounded by the intestinal resonance. This dulness extends somewhat lower during a full inspiration.

In setting limits to the *stomach* and *intestines*, by means of percussion, we have to judge more between sounds of different degree, but similar to one another, than between sounds of different character. Nor are the tones elicited always the same over the same spot; they vary as the contents of the hollow viscera vary. We can make use of this circumstance for purposes of diagnosis. In percussion of the stomach and of the intestines we may often with great advantage resort to auscultatory percussion.

The stomach, when not unusually distended with gas or with food, renders a sound which is hollow, ringing, and tympanitic to a certain degree, yet which is not tympanitic as that of the intestine is. To determine the boundaries of the stomach, it is necessary to mark out first the lower margin of the liver, for it covers a portion of the stomach; then the heart and the inner border of the spleen. The part which lies between these solid viscera yields the sound of the stomach, mixed at one point, namely, to the left of the apex of the heart, with the resonance of the lung. Near this spot, about opposite to the seventh rib, and on the left of the body of the tenth dorsal vertebra, the cardiac extremity of the stomach is situated; below it is the bulk of the organ; the pylorus is on the body of the first lumbar vertebra, four to six centimetres to the right of the median line, on a level with the tip of the xiphoid cartilage, between the right edge of the sternum and a vertical line passing through the nipple. Fully three-fourths of the stomach is to the left of the median line. The cardiac end is immovable; the pylorus, seated from six to eight centimetres lower than the cardiac, has a moderate mobility; in infants it is nearer the median line than in adults.

To ascertain the lower border of the stomach, we percuss gently in a downward direction, until the alteration in sound shows that we are striking over the colon. The difference is at times very obvious, at times very slight. It is readily detected if the stomach contain either solid or liquid ingesta. Availing ourselves of this fact, we may with advantage let the patient swallow a glass of water. By placing him in the erect position, the fluid gravitates to the greater curvature.

FIG. 53.



Results of abdominal percussion, as set forth in the text. The dark shades indicate marked dulness; the light shading exhibits a lessening of the clear or of the tympanitic character of the sound,—an approach to dulness.

and the line of comparative dulness indicates the lower margin of the stomach, which is generally found one to two inches above the umbilicus. In men the lower border of the stomach is a little higher than in women; in working-women it is higher than in other women; in children under fifteen years of age it very rarely extends to the umbilicus; in persons of fifty it is not unusual for it to do so. In strong, healthy people the whole position of the stomach is more horizontal

than in weak ones.[1] Deep percussion is used to limit the superior line, and light percussion the inferior line, of the stomach.

Another method to determine the limits of the organ, as well as whether the pylorus is still capable of self-closure in the direction of the duodenum, or is permanently patent, has been proposed by Epstein. It consists in the distention of the stomach by means of carbonic acid, generated by first letting the patient swallow about a teaspoonful of sodium bicarbonate dissolved in a glass of water and then an equal amount of a like solution of tartaric acid. The same end may be attained by direct insufflation by means of injecting air into the stomach through a tube. The stomach becomes very much distended, and emits a deep tympanitic note on percussion, unlike that over the intestines; but if the pylorus be incapable of closure, the intestines too become swollen, and their tympanitic note is changed.

The *colon* yields, in all its parts, a sound of a purer tympanitic character than the stomach, the note of which is, indeed, in many respects more amphoric than tympanitic. When the tube contains fæces, the sound is modified; and as these are prone to accumulate on the left side in the descending colon, and especially where this passes into the iliac fossa, it is usually not so resonant as the ascending colon. The *small intestines*, unless they are filled with fluid or solids, or distended with gas, render a sound of higher pitch and of smaller volume than the surrounding large intestine, and by the less deep-toned sound their position may be accurately determined. Artificial distention of the colon, by generating carbonic acid in it by means the same as just mentioned passed into the lower bowel, has been advocated for diagnostic purposes by Ziemssen.[2] It enables us to distinguish with ease the outline of the large intestine, and shows whether there is communication with adjacent organs, such as the stomach, the bladder, or the small intestine. Anomalies of position and form of the bowel give rise to differences in the results of abdominal percussion, as has been well shown in a careful clinical study by Curschmann.[3]

The position of the viscera in the pelvis cannot be ascertained by means of percussion. It is only when the bladder is much distended, or the uterus augmented in size, that the outline of either can be traced on the walls of the abdomen.

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[1] Obrastzow, Deutsches Arch. f. klin. Med., Bd. xliii., 1888.

[2] Deutsches Archiv für klinische Medicin, Bd. xxxiii., June, 1883.

[3] Ibid., Bd. liii., June, 1894.

AUSCULTATION.

Auscultation is serviceable in aiding in the detection of an abdominal aneurism; and sometimes an enlarged spleen gives rise to a distinct blowing murmur; or the rubbing of a roughened peritoneum may occasion a friction sound; but, on the whole, the application of the stethoscope to the abdominal walls is rarely of aid except in determining the significance of abdominal pulsation. In health no constant sound is heard save that of the aorta; for the rush of blood through the other arteries, or through the veins, produces no appreciable murmur. When the stomach is distended with air and contains liquid, sounds possessing a metallic character are perceived, which an inexperienced observer is apt to consider as originating in the lungs, over which, in truth, they are often audible. Similar sounds, together with succussion-phenomena may be elicited when gas and liquid are present together in the peritoneal cavity,—as a result of perforative peritonitis. The passage of gas through the intestines gives rise to those peculiar noises termed "borborygmi." In cases of stenosis of the bowel a hissing sound is sometimes audible during peristaltic activity. In the pregnant state, auscultation is of value in detecting the pulsations of the fœtal heart and the utero-placental murmur.

SECTION 1.

DISEASES OF THE STOMACH.

It is only within the last few years that any attempts have been made to bring to bear on the diseases of the stomach modern means of research. Most of these attempts have had as their aim to ascertain the exact anatomical changes and the modifications in the secretions which give rise to the symptoms commonly referred to perverted function: and they have been successful to a decided degree.

The stomach is examined partly by physical exploration by the methods just detailed, and partly by paying attention to the chemical changes which attend the digestive acts.

With reference to the physical examination, there are some special means that may be employed with advantage. To determine the relative sensitiveness over the epigastrium, Boas[1] measures the pressure by an *algesimeter*. The normal tolerance is from eighteen to twenty pounds. In cases of gastric ulcer, pain is complained of at a pressure of from two to four pounds. The direct application of *electricity* to the coats of the stomach as a test of their motility has been also made

[1] Münchener Medicinische Wochenschrift, Sept. 1893.

use of; but, valuable as this agent has proved therapeutically, it has not shown itself valuable diagnostically.

Ingenious instruments have been devised for illuminating and inspecting the interior of the stomach. By means of the *gastrodiaphane* of Einhorn,[1] which consists of a soft rubber tube through which pass wires connected with a source of electricity and provided with an incandescent lamp enclosed securely in glass, the size and outlines of the stomach, as well as the density of its wall, can be made out. The patient is, on an empty stomach, first made to swallow, or there are introduced through a tube, one or two pints of water; the tube is passed into the stomach in the customary manner, and the appearance of the light is observed in a dark room. A reddish luminous zone upon the abdomen indicates the outline and the position of the stomach; and dark spots may enable us to judge accurately of the size, shape, and position of tumors. Gastrodiaphany has also been made use of in the diagnosis of œsophageal diverticula, in which, moreover, the swallowing sounds are frequently audible.[2]

The *gastroscope* of Mikulicz is a more complicated instrument, by means of which it is possible to inspect directly limited portions of the interior of the stomach. A revolving sound, the *gyromele*, has been invented by F. B. Turck.[3] The revolutions can be felt upon the abdominal wall, and the various parts of the stomach, especially the greater curvature, accurately located. If the movements of the sound are distinctly felt on the parietes, tumors of the anterior wall and of the fundus can be excluded.

It is always important to study the activity of the movements of the stomach, and this is generally done partly by noting how long it will take a trial meal to digest completely, partly by chemical means to be presently detailed. But the object has been also sought to be attained by instrumental aid. With this view, Turck[4] has introduced a *gastric motormeter*, which consists of a collapsed rubber bag with a fine rubber tube attached that is connected with a manometer; thus both degree and force of movement are registered. The bag is inflated with air after being passed into the stomach. Another way of determining the mechanical action of the stomach, as well as of recording its movements, is by the *gastrograph*, the invention of Einhorn.[5]

The accurate chemical study of the secretions and of the contents

[1] New York Medical Journal, Dec. 1892.

[2] Jung, Amer. Journ. Med. Sci., April, 1900.

[3] Journal of the American Medical Association, March, 1895.

[4] Proceedings of the American Medical Association, May, 1895.

[5] New York Medical Journal, Sept. 1894.

of the stomach is leading to great advances in the investigation of its affections, as has been proved especially by the labors of Leube, of Ewald, of Boas, and of others. We get the contents of the stomach for examination from two to four hours after a full or "trial meal," given as a mid-day dinner, and consisting of four hundred grammes of soup, sixty grammes of scraped meat, and fifty grammes of white bread: of this, if the act of digestion have been normally carried on and the chyme have passed on into the small intestine, nothing remains after the lapse of six or seven hours but a clear liquid. Ewald has substituted a light breakfast trial meal, a small amount of dry bread or of toast, from thirty-five to seventy grammes, and a third of a litre, about eleven fluidounces, of warm water or weak tea, which, given on an empty stomach, allows the gastric contents to be tested in an hour, a matter often of great convenience. The material for examination is obtained by means of an elastic tube, preferably of soft rubber, about seventy-five centimetres long by six centimetres in diameter, and provided with an opening at its conical extremity and others at the side. The liquid is removed from the stomach by pressure over the epigastrium, or by aspiration by means of a hand ball apparatus. The results of these trial meals should be filtered for accurate examination. When vomiting takes place an examination of the ejecta may yield evidence of the digestive and motor activity of the stomach, or of the presence of abnormal elements.

The next points to determine are the composition of the gastric juice and its digestive power. We first have to ascertain if the liquid obtained be acid, how great its total acidity, and what its acid nature is owing to. The acid of the gastric juice is hydrochloric. Lactic acid plays no part in the normal digestive process. When the latter is present, it is derived from the food, or it may result from the fermentative activity of bacteria. Its presence is indicative of stagnation of the gastric contents or of hydrochloric acid deficiency. The total acidity one hour after an Ewald test breakfast is normally about 60. The average amount of free hydrochloric acid is from 20 to 30, or equal to 0.1 to 0.2 per cent. The best indicator for the total acidity is phenolphthalein.

To determine the presence of free acid in the gastric contents, the most delicate reagent is Congo red, which may be employed in solution or in the form of paper impregnated therewith. Free acid causes an azure-blue color; acid salts have no effect. A solution of methyl-violet may also be employed, which is turned into a deep blue; or tropæolin, which in a saturated watery solution is a dark yellowish-red liquid that on contact with any free acid becomes dark brown, while

with acid salts it assumes a straw-colored tint. To ascertain the presence of hydrochloric acid a good test is Günzberg's phloroglucin-vanillin solution. It consists of two grammes of phloroglucin and one gramme of vanillin, with thirty grammes of absolute alcohol. A few drops of this solution, which is of a yellowish-color, added to a similar quantity of a liquid containing hydrochloric acid, when gently heated, turn it at once a bright-red hue; while the reagent remains unchanged by organic acids, such as lactic or acetic acid. Boas[1] recommends a solution containing resublimated resorcin five grammes, white sugar three grammes, dilute alcohol sufficient to make one hundred grammes. Of this, three or four drops are added to five or six drops of the gastric contents, and the whole is gently heated to dryness; a bright-red hue results from the presence of free hydrochloric acid. The simplest and quickest test is the dimethyl-amido-azo-benzol test of Töpfer, and it is one coming into general use. Both Hemmeter[2] and Stockton[3] regard it as the best. A few drops of a 0.5 per cent. alcoholic solution added to the stomach contents develop a cherry-red color if there be free hydrochloric acid. The acidity referable to this is 35 degrees.[4]

To determine the presence of lactic acid, a matter often of very great value for diagnostic purposes, a solution is prepared of ten cubic centimetres of a four per cent. solution of carbolic acid, twenty cubic centimetres of water, and one or two drops of a solution of ferric chloride. This has an amethyst-blue color, which in the presence of lactic acid becomes lemon-yellow or canary-yellow.

Yet the test is not altogether trustworthy, as sugar, peptone, alcohol, and other substances also cause a yellowish coloration; further, it is interfered with by the presence of phosphates and hydrochloric acid in considerable amount. To remove these sources of possible fallacy, Strauss[5] has recommended the following procedure. Into a graduated funnel are introduced five cubic centimetres of gastric juice and twenty cubic centimetres of ether, and the mixture is vigorously shaken. When the fluids have separated, the lower five cubic centimetres are permitted to escape, and sufficient distilled water is added to make twenty-five cubic centimetres, followed by two drops of a solution made up of one part of ferric chloride and nine parts of distilled water. The mixture is again shaken and the lower watery layer appears of

[1] Diagnostik u. Therapie der Magenkrankheiten, 3. Aufl., 1. Theil, 1894, p. 149.

[2] Diseases of the Stomach, 2d edit., 1900.

[3] System of Practical Medicine by American Authors, vol. iii.

[4] Hemmeter, *ibid.*, p. 165.

[5] Berliner klinische Wochenschrift, 1895, No. 37, cited by Sahli, Lehrb. d. klin. Untersuch., 1899.

a deep yellowish green when more than one per cent. of lactic acid is present.

The presence of volatile fatty acids, butyric acid, acetic acid, etc., in noteworthy amounts, may be recognized by the characteristic odor.

The *degree of acidity* of the gastric juice is more difficult to determine than the presence of the acids. Ewald recommends, as a ready way, to titrate with a one-tenth normal sodium hydroxide solution, ascertaining the saturation point with litmus paper or with phenolphthalein. Töpfer's test is now much employed for the quantitative analysis of the stomach acids, and enables us to estimate not only the amount of free hydrochloric acid, but also the acidity due to the organic acids and the acid salts, as well as to the hydrochloric acid that exists in combination with the albuminous bodies. In Töpfer's method three indicators are used. A few drops of a 0.5 per cent. alcoholic solution of dimethyl-amido-azo-benzol added to ten cubic centimetres of filtered gastric juice are titrated with a decinormal solution of caustic soda until the red color due to the free hydrochloric acid changes to a clear yellow. A few drops of a one per cent. aqueous solution of alizarin added to a second portion of ten cubic centimetres of the gastric juice become, when titrated sufficiently with the solution of caustic soda, clear violet, and the test indicates the amount of free hydrochloric acid, of organic acids, and organic salts. A third portion treated with a one per cent. alcoholic solution of phenolphthalein turns dark red when all the acids, including the combined hydrochloric acid, have been saturated. From these different data the amount of the entire acidity, as well as of the separate acids can be calculated.[1]

We may test the solvent power of the gastric juice by taking a piece of hard-boiled egg and adding the gastric juice in a test-tube. Heated in a culture oven, the egg albumin, if the gastric juice be normal in pepsin, will be dissolved in about three hours. Propeptone and peptone are determined by the biuret reaction. The presence of the *lab-ferment* or rennet-ferment is shown by the coagulation, in from ten to fifteen minutes, of between five and ten cubic centimetres of fresh, unboiled milk of neutral reaction exposed in an incubator to the action of from three to five drops of gastric juice. In the absence of lab-ferment the presence of lab-zymogen is shown by the formation of a dense coagulum within ten or fifteen minutes, when a mixture of equal parts of unboiled milk and gastric juice rendered alkaline by lime-water is placed in an incubator. After an hour from the time

[1] For examples refer to Hemmeter, *loc. cit.*

Ewald's trial breakfast has been taken there should be no reaction for starch found by Lugol's solution in the filtered liquid of digestion. An excess of hydrochloric acid in the gastric juice quickly checks the digestion of starch begun in the mouth by the saliva, while a deficiency permits its completion. Under the first condition, therefore, the reaction for starch will be prolonged; under the latter shortened.

The *absorptive activity* of the gastric mucous membrane is shown by the rapidity with which iodide appears in the saliva after the ingestion of one and one-half grains of potassium iodide carefully enclosed in a gelatin capsule. In health the characteristic blue coloration is, as a rule, obtained with starch-paper in the course of ten or fifteen minutes. This test may be modified so as to indicate the digestive activity of the gastric juice by wrapping the potassium iodide in some impermeable material fastened with strands of fibrin. Disintegration of the fibrin permits of the escape and absorption of the iodide, and the time of appearance of iodine in the saliva is an index both of digestive and absorptive activity.

The *motor activity* of the stomach is determined by the development of a violet color on the addition of a drop or two of a neutral solution of ferric chloride to two or three drops of the urine placed upon bibulous paper, after the ingestion of fifteen grains of salol in gelatin capsules at the height of digestion. The violet color shows the presence of salicyluric acid, which is in the majority of persons observed in the course of from sixty to seventy-five minutes, and does not persist for more than twenty-six or twenty-seven hours. But there are still many clinicians who prefer the older method of examining the contents of the stomach, after trial meals, with a view to determine the gastric motility. Leube's method consists in removing the contents of the organ six or seven hours after a trial dinner, or they may be examined an hour after Ewald's trial breakfast. In either case, the stomach should then contain nothing but the liquid of digestion; two hours after the trial breakfast it should be empty. If more than forty cubic centimetres are obtained an hour after Ewald's trial breakfast, it shows insufficient motor activity.

The symptoms which are constantly met with in derangements of the stomach, whether organic or functional, are loss of appetite, nausea and vomiting, acidity, flatulency, and pain.

Loss of Appetite.—This manifests itself in various ways. It may amount to absolute repugnance to taking any kind of food, or may be merely an inability to partake of certain articles. What the loss of appetite depends on, we do not know. That nervous influence has something to do with the anorexia, is shown by the sudden dep-

rivation of all desire to eat when any strong impression is made on the nervous system,—such as that caused by the unexpected receipt of unwelcome news. The collection of epithelium on the mucous membrane is also connected with a marked diminution of the appetite; for with a tongue much coated, absolute disgust at the mere thought of taking food often exists, which yields to relish for food as soon as the tongue begins to clear.

Attending lost appetite, we meet sometimes with great emaciation and with signs as if even the small quantity of food taken were not absorbed into, or utterly failed to nourish, the system. There is apt to be sensitiveness over the abdomen, and spots of particular sensitiveness exist which correspond to the situation of the mesenteric glands. We find, however, no evidence of organic disease, either in the abdomen or in the lungs; nor does this *pseudo tabes mesenterica*, if I may so call it, occur, like the disease it simulates, in scrofulous or tubercular patients. I have met with a number of cases, chiefly in young women with lowered vital force, fond of excitement, and living indolent lives. Some were hysterical, others not. But in all the complaint seemed to be due to deficient nerve-power, with impaired function of the stomach, and possibly of the abdominal glands. This disorder is probably the same as that described by Gull as hysteric apepsia,[1] and kindred to the one delineated by Lasègue as hysteric anorexia.[2]

Instead of the appetite being lost, it may be capricious, or even ravenous. There is great craving for food in diabetes. A craving for food is not often combined with a structural lesion of the stomach. Yet we occasionally meet with it in persons affected with gastric ulcer. It is common to find it in those who suffer from neuralgia of the stomach. And sometimes in cases of mere nervous gastric disturbance, with or without pain, there is an extraordinary exaggeration of the appetite, a *bulimia:* the patient eats largely eight or even fifteen times a day, digests his food, yet is constantly hungry.

The feeling of *thirst* does not lessen when the desire for food does. On the contrary, it usually increases when the latter diminishes.

Excessive Acidity of the Stomach.—Excessive acidity occurs from various causes. The gastric juice may be secreted in great quantities, or it may contain an abnormal amount of acid. But excessive acidity is far more frequently due to the decomposition of food and to a process of fermentation dependent rather upon an insufficient amount or altered state of the gastric solvent. It then manifests itself only

[1] Transactions of the Clinical Society, vol. vii., 1874.

[2] Archives Générales de Médecine, April, 1873.

after meals. When the mucous membrane is covered with a tenacious mucus or with thick layers of epithelium, slow digestion and acidity from fermentation result; because, although the gastric juice is sufficient, it cannot mix as readily with the aliment.

The acids formed in the stomach are, besides the hydrochloric acid of the gastric juice, lactic acid, acetic acid, carbonic acid, butyric acid, and oxalic acid; all except the hydrochloric acid are the result of decomposition. Some articles of food produce these different acids in considerable quantities. Thus sugar generates large amounts of lactic acid. The mode of detecting these acids, and of establishing whether the extreme acidity is due to excess of hydrochloric acid or to other acids, as tested after a trial meal, has been above explained. In examining for acids, the two acids of greatest value to determine are hydrochloric acid and lactic acid. In determining the acidity of the stomach contents we must first ascertain the whole amount of acidity present in the stomach contents after the trial meal, and then the percentage of hydrochloric acid.

The acids which are created in the stomach may give rise to various disorders. When much acid is present it occasions a sensation of heat which extends along the œsophagus. This "heart-burn" is apt to happen in paroxysms, and is attended with a feeling of constriction or with actual pain at the epigastrium. It simply denotes great acidity, and is common in gouty persons. It probably arises from the action of the acid contents of the organ on the sensitive nerves of the cardia and of the œsophagus, and the acid is mostly owing to fermentative changes. When the acidity is due to increase of hydrochloric acid, from excessive acidity or quantity of the gastric juice, it is the result of a gastric neurosis; there may be acid vomiting coming on irrespective of food, and happening in the night or during the early morning hours. What has been called *gastroxynsis* by Rossbach is a gastric neurosis appearing at intervals mostly after some psychical or mental disturbance, and marked by extremely acid vomiting and headache, like that of migraine.

Flatulency.—The gas in the intestinal canal may be merely air which is swallowed; or it may be generated from imperfectly digested food; or it may be a secretion from the blood-vessels of the part. In those who suffer from indigestion it is produced in the last two ways, and the patient complains greatly of the annoyance it occasions. It causes a disgust for eating, a feeling of distention, and sometimes actual pain. By interfering with the downward movements of the diaphragm it induces a sensation of constriction in the chest, shortened breathing, palpitation of the heart, and the sleep is broken by uneasy dreams.

An expulsion of the gaseous contents of the stomach by the mouth gives rise to *eructation*, or belching. The belching which follows the decomposition of food has sometimes the taste and the odor of sulphuretted hydrogen. At other times the eructation is odorless, because the gases formed are carbonic acid, or hydrogen or nitrogen, or some of their compounds. When the gas results from fermentation or decomposition of food, it frequently coexists with acidity occurring only after meals. When it is a secretion from the blood-vessels it happens in an empty state of the stomach, and is often relieved by avoiding too long intervals between the meals. As a cause of flatulence and eructation which it is important not to overlook may be mentioned thoracic aneurism.[1] Marked flatulency is often only a form of gastric neurosis. It is common in nervous dyspepsia and in hysteria.

Nausea and Vomiting.—These are often combined. But sometimes there is persistent nausea without vomiting; sometimes vomiting occurs without any or with but slight nausea. Yet they are both occasioned in much the same way: what gives rise to one will generally give rise to the other.

Vomiting is a complex act. But its causes, although various, may all be arranged under four heads. It either arises from an irritation of the peripheral extremities of the nerves which supply the parts more directly concerned in the act itself, such as the stomach, the diaphragm, and the œsophagus; or the irritation originates in the centres from which these nerves spring, and is referred to their peripheries; or there is a mechanical obstruction in the stomach or intestines; or the vomiting is purely sympathetic. Under the first head belongs the vomiting observed in acute or chronic inflammation of the stomach, in ulcer, or in cancer; also that following a debauch, or the introduction of irritating substances into the viscus. Under the second head may be ranged the vomiting which occurs in diseases of the brain; perhaps, also, that which arises in morbid states of the blood, as in uræmia. Under the third head we may class the vomiting in narrowing of the œsophagus and of the pyloric or cardiac extremity of the stomach, in hour-glass constriction of the stomach, and in obstructions of the intestine. The fourth group is exemplified by the vomiting in pregnancy, in wounds of the extremities, in inflammation of the peritoneum, of the intestines, and of the liver, in renal calculus, and in irritation of the fauces.

Connected thus with so many various conditions, the act of vomiting, taken by itself, is of little diagnostic value. It presupposes a

[1] Walter F. Atlee, Amer. Journ. Med. Sci., July, 1869.

certain amount of irritation existing in the stomach, or reflected to it; but nothing more. As it is allied to morbid states too numerous to be here examined in detail, I shall content myself with making general statements regarding the indications to be drawn from it.

When vomiting is observed in a person previously in good health, we may suspect either the invasion of some acute malady, or that some poisonous substance has been swallowed. Again, it may come on suddenly from violent mental emotion. When everything that is taken is immediately expelled, the difficulty lies in the œsophagus, or at the cardiac orifice of the stomach, or in an extreme irritability of the viscus; and this irritability, attended as it often is with unceasing nausea, experience proves to be more frequently due to sympathetic excitement of the organ than to structural gastric disease. But speedy vomiting, generally without preceding nausea, is also among the symptoms of visceral hysteria. I have known it associated or alternating with extraordinary flatulency.

Nervous vomiting occurs where there is no lesion in the stomach or irritation of food as the cause. It is mostly due to reflected irritation of the nerve-centres controlling the act of vomiting, and is often found in disorders of the uterus; or arises from direct irritation of the nerve-centres in affections of the brain and cord. It is common in hysterical subjects. It is not associated with nausea, and may be of long duration. It is sometimes a primary gastric neurosis, and as such is seen, particularly in neurasthenics, in association with the condition described by Kussmaul as "peristaltic unrest." This is a very annoying symptom, in which there are loud borborygmi and gurgling, especially after eating. The functional vomiting of hysterics presents the curious feature of nothing apparently being retained on the stomach, yet the patient remaining fairly well nourished. There is no nausea with the vomiting. Cases of the kind are sometimes met with where there is no obvious hysteria, but where overwork and anxiety are the cause.

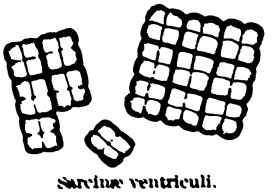
As regards the vomiting which is brought about by gastric disorders, it is of much consequence to note the period at which it happens, whether before meals or after meals, and how long afterwards. In some diseases, such as ulcer and cancer, it rarely occurs except when food has been taken. The act of vomiting then affords relief from the pain. In narrowing of the pylorus, it takes place some hours after digestion has begun. But, as vomiting will be described hereafter in its relations to the individual diseases of the stomach, we shall not dwell on what will be more fitly discussed elsewhere. Yet a few words on the characteristics of the ejected matter can hardly be omitted.

The nature and the quantity of the vomit are, of course, most various. The following are its most common kinds:

Food or *liquid*, mixed with saliva and some mucus, is expelled when the stomach is very irritable, or if an obstruction exist which renders the entrance into the organ difficult or impossible. Half-digested food, in a state of acetous fermentation and with a strongly acid reaction, is cast out when there is deficiency of hydrochloric acid, or when the food has been detained for a long time in the stomach. In the ejected matter the particles of food may be recognized; but when the food has been kept for a prolonged period in the stomach, or when it has passed on into the duodenum and is returned, it is changed into an apparently homogeneous mass. Examined under the microscope, the structures of the animal or vegetable substances partaken of can even then be detected. Mixed with muscular fibre, elastic tissue, starch-corpuscles, and vegetable cells, is found usually a quantity of oil-drops and fat-crystals. The starch corpuscles are turned blue by a solution of iodine and iodide of potassium.

Sarcinæ and yeast fungi are sometimes discovered, by means of the microscope, in the vomit. These organisms are associated with the process of fermentation, and are generally attended with copious vomiting. They are small square or slightly oblong bodies, divided into similar smaller portions by cross-lines, and each portion thus formed is again subdivided; but the markings of the smaller squares are not so distinct as those of the larger. The illustration shows a mass of sarcinæ found in the vomit of a patient who suffered from gastric ulcer.

FIG. 54.



Sarcinæ ventriculi.

Vomit containing sarcinæ is always indicative of structural change in the stomach. It is sometimes found in chronic gastritis of long standing; or in connection with ulcer, and yet oftener with cancer, and especially in those cases in which the narrowing at the pyloric extremity has led to distention of the organ; indeed, any form of dilatation, or a condition preventing the stomach from completely emptying itself, pre-eminently gives rise to it.

Sarcina vomit has an acid smell and reaction, and often a peculiar brownish appearance. After standing, it becomes covered with a dirty, frothy matter, like yeast. A solution of iodine and iodide of potassium turns the sarcinæ mahogany brown or a violet hue; but it is by the microscope that their presence can be recognized with greatest certainty. The process of fermentation attending the develop-

ment of the sarcinæ occasions heart-burn and extreme flatulency, and the copious vomiting is a source of relief.

Mucus is occasionally ejected in large quantities, both mixed with food and pure. In chronic gastritis, and in the milder forms of acute gastritis, the mucous membrane is covered with a tenacious secretion, and a considerable amount of a glairy or stringy matter is expelled by the act of vomiting. As a general rule, indeed, it may be stated that, when much mucus is evacuated, a catarrhal state of the stomach is present.

A thin, watery fluid, looking much like saliva, is discharged in some cases of organic disease of the stomach, as well as in functional derangement of the organ brought on by eating coarse food. Now and then it is met with in pregnancy. This variety of vomiting is known as *pyrosis;* popularly, as "water-brash." It may be attended with a burning sensation extending to the fauces, and with pain running back to the spine. The fluid is commonly alkaline. Frerichs found that it possessed the power of converting starch into sugar. It is mostly regarded as being formed by the glands at the lower part of the œsophagus, while others hold that it is the saliva which has been swallowed and accumulated in the stomach.

Bile may find its way into the stomach, and be expelled by the mouth, imparting to the vomit a greenish or yellowish color and a very bitter taste. The occurrence of bilious vomiting is commonly held to indicate a disease of the liver, or that the patient is extremely "bilious." It is not a proof of either. It is observed when there is much retching, and when the act of vomiting is protracted and frequently repeated, and is chiefly met with in the various forms of acute gastritis, and on the invasion of some acute malady which gives rise to sympathetic gastric disturbance.

Fecal vomiting never depends upon a disease of the stomach. It may be possibly owing to a fistulous opening between the colon and the stomach; but such cases are extremely rare. Generally it is due to a mechanical obstruction to the passage of fæces. Occasionally it happens in fevers of a low type, or in peritonitis, and is then, perhaps, the result of paralysis of a portion of the intestinal tube, which acts, to some extent, as a mechanical obstruction. The matter that is ejected has the odor of fæces; but it is of less firm consistence, and of lighter color, because it is the contents rather of the small than of the large intestine. Sometimes it is perfectly fluid. In fecal vomit a considerable number of large comma-like bacilli have been noticed.[1]

Pus in small amount is sometimes found mixed with the vomit in

[1] Von Jaksch, Klinische Diagnostik.

cases of large ulcers in the stomach, simple or cancerous. When in quantities, it is owing to an abscess in the neighborhood of the viscus having poured its contents into it. Still, pus is rarely met with in the matters expelled. And the same can be said of other substances that find their way into the stomach, like echinococcus sacs and worms, and masses of false membrane.

Blood, on the other hand, is not infrequently vomited. Having described the appearance of the blood when it comes from the stomach, in treating of the diagnosis of hemorrhage from the lungs, I shall, before examining into the circumstances which cause a hæmatemesis, merely here recall the fact that it is preceded by nausea and followed by black stools, and that the fluid ejected is generally black, and presents an acid reaction.

The quantity of blood lost varies greatly; but the amount vomited is by no means a proof of the amount effused. The larger portion may pass off by the bowels, giving rise to peculiar tarry stools. Nay, the whole may be voided with the stools. Chocolate-colored material discharged by stool, and due to alkaline fluids acting on the blood after the effect of acids, is held to be a distinguishing trait between the blood passing by the intestines after a gastric hemorrhage and bleeding from the bowel.[1]

Hemorrhage from the stomach is variously caused. It may spring from injury to the organ, or from disease of its coat; it may be vicarious; it may be the consequence of disorder elsewhere than in the stomach, as of a mechanical obstruction in the portal system; it may depend upon an altered state of the blood.

In the hemorrhage that follows blows or kicks on the stomach, an *active* hyperæmia of the mucous surface is occasioned, which leads to the extravasation of blood. An active arterial hyperæmia also precedes the hemorrhage that sometimes follows the swallowing of irritant poisons. Of organic affections of the stomach only cancer and ulcer are apt to present hemorrhage as a prominent symptom: and of these, again, it is much more frequent in the latter than in the former. The blood effused may be so slight in amount as to escape detection; and this is especially likely to happen when it is intimately admixed with food or with bile. Yet, by means of the microscope, the existence of blood-corpuscles in the ejected matter can be always demonstrated. The fulness of the vessels may be associated with degeneration of their coats, as, for instance, in amyloid degeneration of the stomach.

[1] Bartholow, Practice of Medicine.

When blood has been detained for some time in the stomach, and has become intimately mingled with the acid contents of the organ, it loses entirely its natural appearance. What is termed "coffee-ground vomit" is blood thoroughly intermixed with other substances. It is the result of a comparatively small or gradual hemorrhage, and as this is the kind apt to happen in gastric cancer, it is common in this affection, though by no means limited to it.

Vicarious hemorrhage from the stomach is not infrequent, and especially frequent is that which takes the place of the menses. It is not dangerous. The blood escapes at the time of the normal discharge, and while the bleeding lasts the stomach is slightly tender, and the digestion impaired. But during the intervals there are no signs of disturbance of the functions of the organ, and no pain, both of which are points of importance in distinguishing between loss of blood caused by suppressed menstruation and loss of blood caused by disease of the stomach.

Gastric hemorrhage, dependent upon a state of *passive* congestion brought on by an obstruction to the flow of venous blood, is occasionally seen in organic affections of the heart. But it is much more common as the result of embarrassment of the portal circulation from tumors or from affections of the liver and spleen. It frequently attends, therefore, cirrhosis and enlargement of the spleen, and is often joined to intestinal hemorrhage.

In gastric hemorrhage resulting from changes in the blood the vessels themselves are toneless, and rupture easily or offer no resistance to their altered contents escaping. This kind of hemorrhage is met with in scurvy, in typhus fever, and in yellow fever.

We see thus that blood is vomited from various causes, and that merely from the occurrence of hæmatemesis we can determine nothing definite as to its origin. Yet the symptom—for a symptom it always is—is of serious import, and when taken in connection with others is of great service in diagnosis. We ought, in chronic cases, first to suspect the hemorrhage to be due to some organic disease of the stomach: when there is no other proof of a structural affection of this organ, we turn to the liver, spleen, or heart for its explanation, or examine carefully every part of the abdominal cavity, to see whether or not a tumor is the source of the disorder. If occasioned by none of these conditions, its cause lies probably in altered blood, or in suppressed discharges. The history of the case is indispensable to any induction.

There is, however, one difficulty present in all instances; and that is, to tell whether the ejected blood has found its way into the stomach

and has been subsequently expelled, or whether the hemorrhage is really gastric. The only method to avoid being mistaken is to scrutinize closely the history and the attending phenomena. Blood may be introduced into the stomach by the bursting of an aneurism, or from an ulcerating pancreas; or it may have been swallowed during an attack of epistaxis or of hæmoptysis, or wilfully, to excite sympathy or to escape punishment. A strange result of gastric hemorrhage, first noticed by Graefe, is double-sided incurable amaurosis. In some cases atrophy of the optic nerves has been found. The symptoms and lesions have been attributed to occlusion of retinal vessels.

To return to the more special symptoms of a deranged stomach.

Merycism, or Rumination.—In this condition food that has been swallowed is brought up into the mouth, sometimes by an impulse of the will, but more commonly involuntarily, and remasticated and again swallowed. Rumination is recognized to be purely a neurosis, and may or may not be associated with other gastric disorder.

Regurgitation of fluid or partly digested food may take place in connection with a relaxed condition of the cardiac orifice of the stomach, and, if obstinate, may lead to pronounced derangement of nutrition. This phenomenon is to be distinguished from a similar occurrence that takes place when the œsophagus is the seat of a pouch or diverticulum that empties itself from time to time.

Pain.—Pain in gastric disorders is sometimes slight, at other times violent. It is often rather a feeling of soreness than actual suffering. It may or may not be increased by pressure, and may be augmented or relieved by the taking of food. If persistent, and accompanied by tenderness at the epigastrium, it is almost always linked to a morbid state of the tissues of the viscus. Uneasy sensations, on the other hand, also happen in functional derangement of the organ while the food is being digested, and may be even attended with slight tenderness at the epigastrium. As a rule, pain and soreness dependent on organic disease may be distinguished from pain and soreness that result from functional disorder by noticing the time at which they take place. If they are more severe soon after meals, or when the stomach is full, and worse after a heavy meal than after a light one, especially of a bland substance like milk, they point to a structural affection. If they occur only when the stomach is empty, and are relieved by food, they are indicative of a functional derangement.

Occasionally the stomach is the seat of violent paroxysms of pain. These are at times linked to a chronic organic affection; at others they are apparently connected with a perfectly sound state of the viscus, and coexist with a tendency to neuralgic pains all over the body, or with

hysteria or neurasthenia; or they may appear as the gastric crises of locomotor ataxia; at others they are brought about by some article of food which the stomach does not tolerate or is unable to digest. The disorder is called *gastralgia*, or *gastrodynia;* it is due to a neuralgia of the stomach. When the predisposition to it exists, exposure to cold and damp, a draught of cold water drunk when heated, sudden and violent emotions, or a collection of wind in the alimentary canal, will bring it on. The predisposition is met with in gouty and rheumatic persons, and in those who are debilitated,—in women who are anæmic, and in men who have been exposed to exhausting influences. Then we also find the gastralgia interchanged with other neuralgic or spasmodic affections, giving way to asthma or to angina pectoris, or, on the other hand, occurring in their place. Clifford Allbutt and others have also pointed out a close connection between gastralgia and aortic regurgitation.

The pain varies much in intensity: it is usually severe and agonizing; but it is not permanent; intervals of rest and comfort succeed to the paroxysms of distress. During a violent attack, the skin is cold, the pulse slow, there are frequently nausea, vomiting, sometimes fainting, and often sensations of utter prostration. The seat of the pain is in the epigastrium, immediately beneath the ensiform cartilage, but it radiates both upward and downward, or to the sides. The patient feels as if the coats of the stomach were being violently drawn together, or rent asunder, or rapidly pierced by a sharp instrument. It is sometimes relieved by the recumbent position and by external pressure. But relief depends much on the condition with which the pain is associated. If it be connected with a chronic gastritis or an ulceration, or a cancer, pressure aggravates rather than alleviates it. There is sometimes sensitiveness to the touch in purely nervous gastralgia, and over a considerable part of the stomach; or slight pressure may augment the pain, but firmly compressing the pit of the stomach will diminish it.

It is always important to discriminate between the cases of gastralgia that may be viewed as pure neuroses and those in which the paroxysms of pain are combined with a chronic lesion. We infer that we have to deal with instances of the former, when the attacks occur in those whose impoverished blood or enfeebled health predisposes to neuralgia, and especially if they happen in women laboring under disorders of the uterus or of menstruation, and the attacks increase about the menstrual period, or in persons who suffer from neuralgic pains in other parts of the body. But the broadest line of distinction is drawn by the state of the digestive apparatus during the intervals. The dis-

ordered digestion, the pain after eating, the persistent tenderness at the epigastrium, the nausea and vomiting, and the other symptoms common in morbid alterations of the coats of the stomach, are not seen in pure gastralgia. A sign generally trustworthy is the alleviation following the taking of food, for which, in truth, there may be a craving; and occasionally cases of gastralgia are met with in which the pain occurs early in the mornings, and is very distressing, but is almost immediately eased by a hearty breakfast.

Gastralgia is common where there is an excess of hydrochloric acid in the gastric juice, though Leube states that test meals show, as a rule, but little change. The form of gastralgia which is produced by some article of food that disagrees with the individual is readily distinguished from the other varieties by observing it to be transient, and by noting its cause. The indigestible substance undergoes fermentation in the stomach, and acidity, flatulent distention, and nausea attend the pain, which ceases when the extreme acidity is neutralized by an alkali, or the offending matter is ejected and the gas expelled.

The remarks just made apply also, in the main, to other manifestations of perverted innervation of the stomach, such as hyperæsthesia, with or without persistent vomiting,—forms happening usually in weak or hysterical persons, or where menstruation is disordered,—but which in the present state of our knowledge are still conveniently classed with gastralgia as forms of gastric neuroses.

The nervous filaments, the irritation of which occasions pain in the stomach, whether paroxysmal or not, belong to the vagus; sometimes, perhaps, the distress originates in the branches of the sympathetic that supply the organ. But we must be careful not to ascribe the seat of every pain which is felt between the umbilicus and sternum, or referred there, to the stomach. Diseases of the pleura, of the heart and its covering, affections of the intercostal nerves, abscess of the liver, intestinal disorders, rheumatism of the abdominal muscles, may give rise to pain in the epigastric region. Spasmodic pain like that of gastralgia may be caused by intercostal neuralgia, by intestinal colic, by disorganization of the tissue of the kidney or of the pancreas, and by the passage of gall-stones or of renal or pancreatic calculi. The strictly paroxysmal character of the pain, its seat in the region of the heart or shooting down the left arm, the agitation and distress, the affected breathing, the severity of the symptoms, distinguish gastralgia from *angina pectoris*, and even pseudo-anginas partake of the graver character of the disease. In the passage of gall-stones the great severity of the pain, the attending nausea and vomiting, the subsequent jaundice, are most significant. But there are puzzling cases;

and what makes the diagnosis more difficult is that in persons affected with *gall-stones* gastralgia is not uncommon, and, on the other hand, an attack of biliary colic may seem to be, or is, started by one of indigestion. The localized spots of tenderness, in the course of the affected intercostal nerve, distinguish doubtful cases of *intercostal neuralgia* from gastralgia. Then, too, a galvanic current removes or greatly lessens the pain of the former. The great safeguard always against error is to bear in mind that painful complaints of the stomach may be mistaken for those enumerated, and to ascertain carefully, in cases of epigastric distress, that there is no cause beyond the stomach to account for it. The nearer, in many instances, the pain is to the median line, or, should it occupy this, the more fixed and confined to a small spot, the greater is the probability of its being dependent upon gastric disease; and pain of the character alluded to is generally indicative of serious malady.

Pain is the last of the symptoms directly referable to the derangement of the viscus itself to which we shall advert. But when the stomach is disordered, other organs also suffer, either through sympathy, or because the irritation is transmitted to them. The bowels are usually in a sluggish condition; it is commonly only when the gastric acidity is extreme that they are relaxed. The viscera within the chest are frequently disturbed. The patient is annoyed by palpitation and shortness of breath after meals; and as he feels the agitation of his heart, and finds that always, after he has eaten, his face is flushed, the palms of his hands are hot, and his temporal arteries throbbing, he is apt to fancy himself laboring under a serious cardiac affection. A dry cough, also, is a not unusual concomitant; but a cough may be the result of coexisting catarrh of the bronchial mucous membrane, or of disease of the lung-structure; and sometimes the affection of the lungs precedes that of the stomach. Again, we may have an organic disease of the heart leading to the gastric symptoms.

So, too, with the kidneys. They may be irritated by the crude material which has made its way into the blood, and which they are called upon to excrete. The urine often contains various abnormal constituents, especially quantities of urates and oxalates. But, on the other hand, a morbid state of the urine may precede the derangement of the stomach, and the indigestion be the secondary rather than the primary ailment. Indeed, we must never be too hasty in concluding, when a disordered stomach is associated with diseases of other viscera, that it is their cause; it may exist as their consequence. Diseases of the liver and intestines are especially prone to induce a gastric affection.

One of the worst results of a disordered digestion is the state of mind it produces. It occasions listlessness and a disposition to look at all events in a gloomy light, and sometimes brings on inveterate hypochondriasis. Aretæus ascribed to the stomach as its primary power that it acted as the president of pleasure and of disgust, "being, from the sympathy of the soul, an important neighbor to the heart for imparting good or bad spirits." Now, although no one at present would agree with this physiology, who will deny that there is in the remark a germ of truth? But here, again, we must be careful not to confound cause with effect; for want of activity or a distressed state of mind may seriously impair the appetite and subvert the normal action of the viscus.

When the nervous symptoms are marked, the disorder is often called *nervous dyspepsia*. In this, while the gastric symptoms may be light, we may also have the gastric neurosis leading to extreme acidity of the gastric juice, to bad taste in the mouth, increased salivation, perverted appetite, to eructations, to flatulency. There may be sensations of distress and uneasiness during the digestive act, and general sensitiveness in the epigastric region, but the gastric motility, contrary to what might be supposed, is not impaired, and the trial meals are digested in their usual time. There is not always increased acidity of the gastric juice. The hydrochloric acid may be normal or diminished in amount. In all forms there are uneasy feelings after meals and great nervousness. Headache, general lassitude, low spirits, at times vertigo and palpitation, are complained of. Nervous dyspepsia is common in neurasthenics and in hysterics. The exact state of the stomach that coexists can be determined only by chemical investigation of the gastric secretion. Leube[1] maintains that the nervous symptoms are induced, because the nervous system itself is in a very irritable state, and produces morbid digestion. Viewed in this light, nervous dyspepsia is a neurosis, and it is explained how it may become complicated with other gastric neuroses, such as gastralgia. But, however produced, its manifestations are evoked by the digestive act.

In the sketch just finished of the symptoms encountered in gastric disorders, no attempt has been made to separate strictly the signs which belong particularly to alteration of its coats from those which occur in mere derangement of its functions,—in other words, I have not tried to dissociate the symptoms of so-called "dyspepsia" from those of actual lesions. And this for two reasons: in the first place,

[1] Diagnose der inneren Krankheiten, 1898, vol. i.

the most palpable indications of organic disease of the stomach are those of disordered function; and secondly, there are no symptoms which belong exclusively to functional indigestion. Nor is it possible to present anything like a complete picture of merely functional, or, as it is still called by some, *atonic dyspepsia;* the combinations are too infinitely varied.

The stomach may be the seat of various neurotic disturbances, some of which have already been discussed. Its motor activity may be deranged in the direction of either excess or deficiency, and resulting, on the one hand, in premature propulsion of the chyme into the intestine, in the development of borborygmi and gurgling or of eructations, in regurgitation or vomiting of food, in rumination or merycism, in spasm of cardia or pylorus; and, on the other hand, in atony or insufficiency of the cardia or the pylorus. Secretory activity may undergo quantitative or qualitative alteration. Finally, there may result a condition of hyperæsthesia or gastralgia, or abnormalities of appetite.

Diseases of the Stomach in which Pain and Soreness at the Epigastrium, and Vomiting, occur.

After what has been premised, it is obvious that the structural diseases of the stomach present but few symptoms that can be regarded as at all characteristic. Indeed, the only ones which can lay any claim to be so considered—and we have already seen that this claim is not always valid—are pain and soreness at the epigastrium, and vomiting. We may, then, take these symptoms as a starting-point in diagnosis, and describe the individual organic affections in which they chiefly occur, speaking first of the acute.

Acute Gastritis.—Inflammation of the stomach may be of varying degree and extent. It may involve only the mucous coat, or the other tunics as well. The condition arises most commonly from the ingestion of food improper in quantity or in quality. Aggravated forms of the disorder may result from the introduction into the stomach of poisons, such as alcohol, the mineral acids, caustic alkalies, or other corrosive substances. The presence of low forms of vegetable life may be an exciting cause, and sometimes the affection is part of a more general process, as of diphtheria, pneumonia, typhoid fever, smallpox, and rheumatism or gout. In rare instances the disease is phlegmonous or suppurative. The severity of the symptoms varies with the character and intensity of the morbid changes. There may be merely redness and thickening of the mucous membrane, with infiltration of the other coats of the stomach; or there may be desqua-

mation, or the formation of false membrane; or, finally, suppuration, necrosis, and ulceration. Among the usual symptoms are anorexia, nausea, vomiting, pain in swallowing, epigastric distress and burning, with tenderness on pressure, usually diarrhœa, though there may be constipation. Thirst, headache, and vertigo also are common, and we may find elevation of temperature, generally not over 102°, with acceleration of pulse, and hiccough, and increased frequency of respiration. In severe cases, symptoms of collapse are met with. The milder cases terminate in recovery, or pass into chronic gastric catarrh. The more severe cases may lead to ulceration or to perforation or to rupture of the stomach, to hemorrhage, or to cicatricial narrowing. In *phlegmonous gastritis*, of which diffuse inflammation of the stomach wall with purulent infiltration is the more common form, there is sudden onset as well as a sense of burning and violent epigastric pain, and vomiting, tenderness, and a feeling of resistance in the epigastric region, and fever. Slight jaundice may also be present, and bilious vomiting; the vomited matter may contain pus. Peritonitis and signs of collapse are apt to follow.[1] The disease is generally primary and the infection direct, but it may be secondary or metastatic. There are very severe cases of ordinary acute gastritis, involving also the muscular coat, which are undistinguishable except by the absence of peritonitis and the fact that they may recover. I have seen such instances. *Membranous gastritis*, a form of gastritis more common in children than in adults, is not to be recognized from any other kind of severe gastritis, unless shreds of membrane and casts are vomited.

A mild gastritis is very commonly brought on by a debauch or by the introduction of irritating articles of diet into the stomach. These cases are classed as *acute gastric catarrh*, and are popularly known as severe attacks of indigestion; that they are owing to an inflammatory state of the mucous membrane was proved by the ocular demonstration Beaumont had of the process in the person of Alexis St. Martin. There is some tenderness at the epigastrium; nausea: vomiting; constipation, or sometimes diarrhœa; a coated tongue, and headache.

Another common and kindred kind of mild inflammation of the stomach or acute gastric catarrh is that usually called a "bilious attack." The French designate it expressively as *embarras gastrique*. It is a catarrhal affection, and may be associated with catarrh of other

[1] See an excellent analysis of the recorded cases by Leith, in the Edinburgh Hospital Reports, vol. iv., 1896.

mucous membranes. It may come on from indigestible food, or after cold and exposure; it sometimes occurs in epidemics. The symptoms are those already detailed. There is nausea, and frequently bile is vomited. We do not usually observe much pain in the epigastrium; but rather a feeling of uneasiness, and a slight soreness to the touch. The urine is dark and deposits urates; the tongue is much coated; there is thirst, with generally a moderate or slight fever, which exacerbates at night, and is of remittent type, and there may be a yellowish tinge of the conjunctivæ. In children acute gastric catarrh may become complicated with convulsions, or with symptoms simulating those of meningitis.

A form of gastritis is described which occurs in very young children and leads to softening of the mucous lining of the stomach, a *gastromalacia*. This softening is most likely a post-mortem change due to the action of the gastric juice, and especially met with in the subjects of acute gastric catarrh. Kundrat has called attention to the occurrence of gastric softening with vomiting of blood in the brain affections of children, especially in tubercular meningitis.

Chronic Diseases attended with Pain, Epigastric Tenderness, and Vomiting.

The chronic diseases of the stomach, like the acute, may be considered in accordance with the pain, the soreness at the epigastrium, and the vomiting that attend them. At all events, they are the symptoms common to the chronic diseases which are susceptible of accurate diagnosis. In these chronic diseases vomiting is found to be a symptom of greater diagnostic value than in the acute,—not the act itself, but the appearances of the ejected matter. Further, the phenomena of dyspepsia stand forth much more conspicuously.

Chronic Gastritis.—In chronic inflammation of the mucous membrane, or *chronic gastric catarrh*, the symptoms of indigestion are persistent and manifold. They vary somewhat according to the extent of the mucous surface involved and the amount of mucus and epithelium which accumulates on it, and also according to the healthy or wasted state of the gastric glands. Generally there is a sensation of discomfort, of weight, and of soreness at the pit of the stomach, aggravated by food; the part is also tender to the touch. Sometimes, even when the stomach is empty, a burning at the epigastrium and an inward fever are complained of. The appetite is impaired or capricious. Fermentation, heart-burn, and flatulency frequently attend the slow digestion of the food; the tongue is usually heavily coated; it may, however, be clean. The bowels are constipated. The urine contains

an excess of urates or of phosphates, or exhibits crystals of oxalate of lime. The patient's circulation is languid; he suffers from chilliness; his spirits are depressed. Not infrequently he is annoyed by thirst, and vomits, after meals, the half-digested food mixed with strings of mucus. But the vomiting may also take place when the stomach is empty, and the ejected matter is then fluid and colorless. Drunkards who suffer from chronic gastritis often throw up a quantity of glairy fluid on rising in the morning. A colorless vomit, joined to symptoms of long-continued indigestion, is very characteristic of chronic gastritis.

The gastric contents removed after a trial meal show a diminution in the amount of hydrochloric acid present, usually in the total acidity also, and in the activity of the digestive ferments; still, hydrochloric acid is generally present. Absorption from the stomach is retarded, although gastric motility is little if at all impaired. The fasting stomach may be empty or contain mucus.

Chronic gastric catarrh may involve the mucous membrane or also the other coats of the stomach. The mucosa may be thickened or it may be thinned; it may be the seat of erosions. The glandular structure may undergo varying degrees of atrophy. All of the coats of the stomach may eventually become sclerotic. When atrophy of the gastric tubules has taken place there is complete absence of hydrochloric acid and of the digestive ferments.

Thus, then, the results of chemical examination of the removed gastric contents, the character of the vomit occasionally, more frequently the coated tongue, the distress after eating, the soreness at the epigastrium, and the persistency of the symptoms, distinguish the dyspepsia of chronic inflammation of the stomach from that which is purely functional.

The causes of the malady are at times obscure. It certainly cannot be traced often to an antecedent acute attack, although those who suffer from the chronic disorder are particularly prone to acute exacerbations. It is more common in persons over than in those under forty years of age. It is especially common in gourmands and drunkards, and in those who live on coarse food or who eat irregularly. It is often found conjoined with chronic bronchitis, with anæmia, with Bright's disease, with tubercular disease of the lungs, with gout, and with diabetes. Passive congestion undoubtedly acts as a predisposing element, and thus originates the chronic gastric catarrh met with in affections of the heart and of the liver.

Chronic gastritis is frequently associated with ulcers in the organ or with cancer, and many of the symptoms of these disorders are

clearly attributable to it. Let us inquire whether there are any special symptoms to inform us that something more dangerous than chronic inflammation of the mucous membrane of the stomach exists.

Gastric Ulcer.—Ulcer of the stomach is a disease comparatively rare in this country; but it is not so in some parts of the Continent of Europe and in England. It seems to be more common in northern than in southern climates. The affection is essentially dependent upon disturbance of the normal relation between the gastric secretion and the circulating blood, in that the one is unduly active and the other is deteriorated in quality. It is more common in females than in males, and between twenty and forty years of age than at any other period. It is generally associated with anæmia, or follows chronic gastric catarrh, or embolic plugging of small arterial twigs, or other disturbances of the circulation in the gastric mucous membrane. Amyloid degeneration of the finer vessels, too, occasions these perforating ulcers. The acid gastric juice acts readily and destructively on the weakened tissues. Rarely, gastric ulceration is due to tuberculosis and to syphilis.

The ulcer or ulcers, for there are sometimes several present, are seated usually on the posterior wall of the stomach, in or near the lesser curvature and towards the pyloric extremity. The great danger arises from perforation of the coats and subsequent peritonitis. But the ulceration may prove fatal by opening a large blood-vessel. Again, the formation of a gastro-colic or a gastro-pulmonary fistula may lead to death; or the protracted suffering and excessive vomiting may gradually exhaust the vital energies. On the other hand, the ulcers may heal by cicatrization; and this, William Brinton tells us, takes place in about half the instances. They may thus form tumor-like masses, or when situated at the pylorus, they may cause obstruction to the passage of the chyme into the duodenum. Perforation, Welch states, happens in about six and a half per cent. of all cases. Recurrence of the gastric ulcer is not uncommon.

In cases which may be regarded as typical, the malady is announced by symptoms exactly like those witnessed in chronic gastritis,—the same uneasiness and pain at the epigastrium, and occasional nausea and vomiting of food, or of a watery fluid. Perforation may at this early stage of the disease most unexpectedly cut short the patient's life. Should perforation not take place, hemorrhage from the stomach, with emaciation and anæmia, next appears. In this way the disease usually continues for months or years, the symptoms remitting from time to time, and showing singular variations in their

severity. Welch[1] states the average duration of gastric ulcer to be from three to five years. The majority of the cases recover.

Of the symptoms, pain and vomiting are the most characteristic. Pain is rarely absent; never, perhaps, except in cases which run a rapid course. It is generally a continuous dull feeling; sometimes a burning, at other times a gnawing sensation. As a rule, it is rendered more acute within a quarter of an hour after eating, and remains so as long as food occupies the stomach. Its situation is commonly in the middle of the epigastric region, and there it continues strictly limited. At this point, too, there is localized soreness, or even great tenderness to the touch. Sometimes the pain is seated behind the ensiform cartilage, or is referred to the right or to the left hypochondrium. It is often associated with a gnawing pain in the lower dorsal vertebræ, which may shoot between the scapulæ or down the spine; but the dorsal pain, like the epigastric, is, on the whole, very fixed, radiates but little, and is most severe when the ulcer is on the posterior surface. Besides this continued feeling of distress, there occur violent paroxysms of pain, which may last for several hours; nay, with trifling intermissions, for days. They sometimes come on suddenly when the viscus is empty, but are aggravated by pressure or by food; and, in fact, they are often thus induced. The patient refers the suffering chiefly to the pit of the stomach, or to the dorsal vertebræ. He is apt to seek the recumbent posture for its relief. Yet it is remarkable that there are at times long intervals during which all pain, whether paroxysmal or not, ceases, and during which food can be taken without inconvenience. The acidity of the urine is diminished; the reaction may even be alkaline; the chlorides are diminished or absent.

The peculiarities the pain exhibits form, on the whole, the most distinctive symptom of gastric ulceration. The paroxysms just spoken of may be mistaken for a purely nervous gastralgia. Indeed, when it is considered that both disorders are specially apt to occur in anæmic women, and in those whose menstrual functions are deranged, it becomes apparent how easily this mistake may be committed. The soreness at the epigastrium; the persistent symptoms of indigestion; the excess of hydrochloric acid in the gastric juice; the increase of pain after meals,—constitute, in a diagnostic point of view, the safeguard against error. To these might be added the vomiting of blood, were it not that vicarious hemorrhages are not at all unlikely to take place in young women who are troubled with amenorrhœa. This is, in truth, a matter having a close connection with the diagnosis of

[1] Pepper's System of Practical Medicine, article "Simple Ulcer of the Stomach."

gastric ulceration. Persons who suffer from disturbance of the menstrual function are prone to be hysterical; and it may happen that one of the most marked traits of the hysterical disorder is that it manifests itself by tenderness in the epigastric region, and by pain in the stomach.

We thus may have the most significant signs of gastric ulcer, occurring, as so many cases of amenorrhœa do, in chlorotic young women; therefore happening in the class among whom ulceration of the stomach is most frequent. Nay, the very history may point to the probability of gastric ulcer.[1] Yet, generally, by close attention to all the phenomena of the case, we can arrive at a correct conclusion. The tenderness, as in all local hysterical affections, is great on the slightest touch; and there is no severe pain posteriorly corresponding to the spot of soreness in the epigastric region. Pressure upon a spinous process may cause pain, but it is not the peculiar dorsal pain of gastric ulceration. Then, in the hysterical complaint there is often hyperæsthesia of the skin in various portions of the body, and the apparent gastric distress bears no relation to the taking of food, or to the circumstance of its being of an irritating character or otherwise. The epigastric surface temperature is elevated in gastric ulcer, and may even exceed the temperature in the axilla.[2]

But to return to the vomiting of blood. When this is not traceable to a suppression of a natural discharge, and when it does not befall a person who suffers from disease of the heart, or liver, or spleen, or œsophagus, it acquires great significance. It is the only kind of vomit at all distinctive of a gastric ulcer; for the substances ejected present otherwise appearances not different from what they do in chronic gastritis. The blood may be pure and red, but it is more frequently blackened by the gastric juice; and large quantities are sometimes passed by stool. Now, hemorrhage does not take place in chronic inflammation of the mucous membrane of the stomach, except perhaps in drunkards, or where there is coexisting disease of the liver or spleen. In those instances in which erosions exist on the surface, the vomited mucus may be a little streaked with blood; yet anything like a profuse hemorrhage never happens. Hence its occurrence in a case with the symptoms of chronic gastritis, cancer being excluded, renders the presence of an ulcer probable. Yet there is a source of fallacy, as I know by having met with such an instance, due to removal of the ovaries in an hysterical woman with marked gastric symptoms,

[1] Case under my care, Philadelphia Hospital; Medical and Surgical Reporter, Feb. 1863.

[2] Hayem, Revue des Sciences Médicales, Oct. 15, 1888.

in whose case subsequent hæmatemesis repeatedly occurred. It must also be borne in mind that we may have gastric ulceration without hæmatemesis, and that in pure hysteria blood may be vomited.

The vomiting of the matters taken into the stomach may be immediate, or not for some time after the food has been swallowed. Usually it happens speedily, and in some instances so speedily that there seems to be rather regurgitation than vomiting. But this is rare, and in the rarity is a safeguard against confounding gastric ulcer with the vomiting of cerebral disease, especially tumor, which I have known to happen in a young woman in whom, moreover, vomiting of blood had occurred. In the regurgitation, then, in the frequently absent nausea, in the clean tongue,—though coating may also be absent in ulcer,—in the want of oppression and weight at the epigastrium, and in the headache, altered vision, and other nervous phenomena, we have the distinguishing traits between *gastric* and *cerebral vomiting* on which to lay stress in the diagnosis between disease of the brain and gastric ulcer, or indeed any other serious stomach affection. The attacks of gastric pain that occur in the *gastric crises* of locomotor ataxia may be misleading. But the absence of knee-jerks and the eye-phenomena explain their meaning. Constipation is present in the large majority of cases of gastric ulcer. Pallor also is a common manifestation. The number of red blood-corpuscles usually undergoes moderate diminution, while the percentage of hæmoglobin suffers a somewhat greater reduction.

Perforating gastric ulcer may lead to localized abscess in different situations near the stomach, and this abscess may burst into the peritoneum, or be discharged externally, recovery ensuing. In some instances the abscess forms beneath the diaphragm, and may be mistaken for pneumothorax. Indeed, this *pyopneumothorax subphrenicus* may show physical signs like those of pneumothorax. But it does not extend to the summit of the chest, and there is but little displacement of the heart. Moreover, the history points to long-existing gastric derangement. Pain in the front of the chest or in the abdomen, as the cases of Penrose and Dickinson[1] prove, is an early symptom, and is soon followed by the physical signs of pneumothorax or of pneumonia.

In concluding this sketch of gastric ulceration, two questions arise which require solution: Does an ulcer always produce the peculiar train of symptoms mentioned? May not the same phenomena be met with in other disorders? The first question must be answered in

[1] Clinical Society's Transactions, vol. xxvi., 1893.

the negative. Ulceration of the stomach may occasion nothing but the symptoms of chronic gastritis; and even these may not be marked. The second question is to be answered in the affirmative. There is a disorder with symptoms almost identical with those of gastric ulcer, the corrosive *ulcer of the duodenum.* Now, this affection, were it more frequent, would be a constant source of error. It may run an acute, or at least an apparently acute, or a chronic course. In either case it is scarcely distinguishable from gastric ulceration. Trier, from an analysis of twenty-six cases, mentions, among the most important grounds for a differential diagnosis, a sensitive tumor in the epigastrium, proceeding from adhesion with the pancreas, and jaundice or other hepatic phenomena. But these symptoms are far from constant; and in acute cases, and in those chronic cases which run a latent course, the diagnosis is impossible. It may be added that the perforating ulcer of the duodenum is much more apt than ulcer of the stomach to remain latent and to lead rapidly to a fatal termination. The most certain signs of duodenal ulcer are the sudden and apparently causeless occurrence of intestinal hemorrhage, which may recur and be associated with hæmatemesis; violent attacks of pain referred to the right hypochondrium or the epigastrium; pain in the right hypochondriac region happening two or three hours after meals; dyspeptic symptoms, generally of moderate degree, and diarrhœa. Duodenal ulcer is thought by some to be almost invariably due to the action of a highly acid gastric juice, and to furnish the best illustration of the so-called "peptic ulcer." It sometimes follows burns of the cutaneous surface. It is most common between thirty and forty years of age, and, as Krauss proves, is ten times more common in men than in women.

Where perforation occurs from duodenal ulcer the symptoms are the same as in perforation from gastric ulcer: sudden, agonizing pain, epigastric first, then becoming diffused; symptoms of collapse, subnormal temperature, rapid breathing, and vomiting, which soon ceases in the case of perforating gastric ulcer, but continues in duodenal ulcer.

There is yet another affection with symptoms like those of ulcer, an affection still more serious and destructive,—cancer.

Gastric Cancer.—Cancer is found more frequently in the stomach than in any other organ except the uterus. Of nine thousand one hundred and eighteen cases of cancer which occurred in Paris from 1837 to 1840, two thousand three hundred and three were in the stomach.[1] Among thirty thousand cases analyzed by Welch the stom-

[1] Walshe on Cancer.

ach was involved in 21.4 per cent. The disease is generally primary. It is most often seated at the pylorus; next in frequency stands the lesser curvature; then the cardiac orifice and the posterior wall; most rarely does it involve the whole viscus. We find all the varieties of cancer affecting the stomach: medullary, adenomatous, scirrhous, colloid, squamous. There may be nodular tumors of varying consistency or more or less diffuse infiltration of the coats of the stomach. Breaking down of the growth may result in the formation of ulcers; and perforation may take place. Occasionally carcinoma develops in the site of a previous ulcer. As found by an analysis of two thousand and thirty-eight cases of gastric cancer, three-fourths occur between forty and seventy years of age.[1] Males suffer more commonly than females, and whites far more than blacks.

The symptoms of cancer of the stomach are the same as those of chronic gastritis,—pain, tenderness in the epigastrium, disordered digestion, vomiting. In a more advanced state of the cancerous malady there may be those of gastric ulcer, hemorrhage being added to the list above given. There is only one symptom distinctive of cancer,—namely, the existence of a tumor.

But let us see if there be anything in the pain and vomiting, or in the circumstances of the case, by which, even when a tumor cannot be discovered, the presence of a cancer may be suspected. Pain is a very constant symptom; quite as constant as in gastric ulcer. But the pain is, as a rule, more continuous, much less influenced by the taking of food, and more radiating, being often referred to the right or the left hypochondrium. Its character is very varying. It may be dull, or gnawing, or it may be lancinating. It may be slight, or it may amount to excruciating agony. But it is a mistake to suppose that a cancer of the stomach necessarily causes severe or lancinating pain. Again, it should be borne in mind that the part diseased may ulcerate, and then the pain is exactly like that of an ordinary gastric ulcer, and is affected in the same way by food. The most marked seat of the pain is sometimes under the shoulder-blade.

Vomiting is not an invariable result of cancer; yet it is a frequent one. The seat of the morbid growth determines, to a great extent, the occurrence of vomiting and the period at which it will happen. When the body of the stomach is attacked, and the orifices are not obstructed, it may not take place at all; or, if it take place, it is within a brief time after meals. When the disease has narrowed the cardiac extremity, vomiting supervenes almost immediately; the food

[1] Welch, Pepper's System of Practical Medicine.

has hardly been swallowed before it is brought up again. But when, as is much more common, the pylorus is constricted, the food is not thrown off until it attempts to pass through into the intestine; therefore not until a considerable time after meals.

With respect to the character of the substances ejected, this too depends on the seat of the cancer, and the time at which the vomiting occurs. If it ensue several hours after meals, the cast-off matter consists of food partly digested, partly in a state of highly acetous fermentation. An enormous quantity of acid material, the accumulation of several meals, is sometimes brought up during one act of emesis. The ejected matter may be intermingled with blood, and have a blackish or reddish-brown, "coffee-ground" appearance; or the mucus which is thrown up may be tinged with black flakes: in either case we find reduced hæmatin. Rarely is any considerable amount of unmixed blood vomited.

Free hydrochloric acid is often absent from the vomited contents of the stomach or from the "trial meal," especially in cancer of the pylorus. But we must not forget that it is also absent in amyloid degeneration, in simple gastric achylia and in atrophy of the gastric tubules, in many fevers, and occasionally in chronic gastritis. The persistent presence of free hydrochloric acid renders the existence of carcinoma very improbable.

It is at times a very difficult diagnosis between cancer of the stomach in which no tumor can be found and *achylia gastrica*. This absence of secretion of the gastric juice shows persistent loss of hydrochloric acid and of ferments, and is found as a primary secretory debility, especially in neurasthenics. But a graver form is associated with atrophy of the gastric tubules, and it is in this affection that, irrespective of the chemical signs, the marked dyspeptic symptoms, the progressive debility and anæmia, and the severe gastralgia make us think of cancer. Vomiting, however, is not a prominent symptom, and, unlike cancer, diarrhœa is.

In many cases of carcinoma of the stomach, lactic acid is to be found in the gastric contents after the administration of a special trial meal, free from lactic acid and lactates, and consisting of oatmeal gruel (a tablespoonful of oatmeal to a quart of water) with a little salt.[1] This phenomenon is rare under other conditions, and though not pathognomonic of gastric cancer, when existing with dyspeptic symptoms and absence of hydrochloric acid, it is almost conclusive. Microscopic examination may disclose the presence in the cancerous particles

[1] Boas, Münchener Medicinische Wochenschrift, 1893, No. 43, p. 805.

found in the gastric contents or the washwater of large numbers of cells showing mitosis, and of characteristic "concentrically arranged conglomerations of cells;"[1] also of unusually long, non-motile bacilli.[2] These bacilli have the power of forming lactic acid freely. They are not pathognomonic of cancer, since they have been met with also in simple hypertrophic stenosis of the pylorus, but they are very important and significant. The Oppler bacillus existed in nineteen out of twenty cases of gastric cancer examined by Kaufmann. In gastric carcinoma, further, the motility of the stomach is generally much impaired, the ferments are defective or absent.

A close study of the pain and vomiting may furnish evidence by which the existence of a gastric cancer may be strongly suspected. There are a few other circumstances which would strengthen this suspicion: such as the sour eructations, the extreme flatulency, the persistent fetid breath, obstinate constipation, anorexia with progressive loss of flesh, and the cachetic appearance of the patient, who is pale and tired-looking, or whose face is of a color which seems to have arisen from a combination of the hue of chlorosis and that of jaundice. The supposed characteristic straw color of cancer is not often met with. The temperature is generally below the norm; but there are exceptional cases in which a moderate amount of irritative fever accompanies the gradual wasting. Œdema of the ankles is a frequent symptom of the advancing disease. In some instances coma happens similar to diabetic coma, or tetany, as Kussmaul pointed out. There is a form in which rapid enlargement of the liver, some fever, and erythematous eruptions occur.[3] The blood presents scarcely distinctive changes. The number of red corpuscles is usually diminished, and the percentage of hæmoglobin in yet greater degree. The number of white corpuscles may be somewhat increased, with an absence of digestion-leucocytosis. Acetone and peptone have been found in the urine.

Now, should all these symptoms be met with in a person who is steadily becoming feebler, whose age is above forty, in whose family cancer is hereditary; should cancerous tumors develop themselves in any other part of the body,—the suspicion entertained would be converted into a certainty. But it is not often that a case presenting a combination of all the symptoms enumerated is met with. I repeat, the most distinctive sign is a tumor: when this is not detected, uncertainty hangs over any diagnosis of gastric cancer.

[1] Ewald, Klinik der Verdauungskrankheiten, 3. Aufl., 2. Band, p. 342.

[2] Oppler, Deutsche Medicinische Wochenschrift, 1895, No. 5.

[3] Hanot, Archives Générales de Médecine, Sept. 1892.

To contrast, then, cancer of the stomach with chronic gastritis and gastric ulcer:

Chronic Gastritis.	Gastric Ulcer.	Gastric Cancer.
Pain at the epigastrium somewhat augmented by food; also soreness. Both constant, although comparatively slight.	Pain at the epigastrium much augmented by food; subsides when this is digested; paroxysms of pain, a strictly localized soreness to the touch in the epigastric region, sometimes a painful spot over the lower dorsal vertebræ. Intermissions in the pain of considerable length are frequent.	Pain frequently of a radiating kind, often paroxysmal, not unusually severe and lancinating, but not of necessity associated with soreness; little or not at all affected by food. Pain rarely remits; never intermits for any considerable time.
Tongue usually heavily coated; may be clean.	Tongue dry, red, streaked in middle; or smooth and moist or slightly coated.	Tongue pale and thickly coated.
Acid eructations.	Eructations occur, are not acid.	Fetid eructations.
Symptoms of indigestion marked.	Symptoms of indigestion sometimes very slight.	Symptoms of indigestion marked. Anorexia; extremely sour stomach.
Sometimes vomiting; especially morning vomiting in alcoholic cases.	Vomiting usually immediately or soon after taking food; often an early symptom.	Vomiting a very frequent symptom; occurs sometimes on an empty stomach; usually preceded by other symptoms.
No hemorrhage, or but trifling hemorrhage; at most, blood-streaks in vomited matter.	Abundant hemorrhage from the stomach common.	Hemorrhage not very abundant, but occasioning frequently coffee-ground looking vomit.
Bowels constipated.	Bowels may or may not be constipated; usually are.	Bowels obstinately constipated.
No fever.	No fever.	Intercurrent attacks of slight fever may occur; but temperature often subnormal.
Not much emaciation; no cachectic appearance.	Frequently extreme pallor and debility.	Progressive loss of flesh, and cachexia; at times hypertrophy of the peripheral lymphatic glands, especially above the clavicles.
Not confined to any age. More common in middle-aged or elderly people. Common in alcoholics.	May occur in middle-aged persons; but is most frequently seen in young adults, especially in young women.	Most common in elderly people; rarely occurs in persons under forty years of age.
Disease may be relieved or cured; is often of very long duration.	Duration uncertain; may get well, may run on rapidly to perforation; on the other hand, may last for years.	Average duration one year; may be shorter; is seldom longer; very rarely reaches two years.
No tumor.	No tumor.	Generally a tumor.
Contents of stomach contain generally free hydrochloric acid.	Hydrochloric acid in excess in contents of stomach.	As a rule, no hydrochloric acid in contents of stomach; often lactic acid present.
No dropsy.	No dropsy.	Œdema of ankles often met with.

The differences laid down in the table are derived from an analysis of well-marked cases. In the early stages of the cancerous malady, a differential diagnosis is impossible. Subsequently, as already stated, the detection of a tumor plays an important part in any deduction. But this remark does not apply to cases of cancer of the cardiac orifice, which are rare, and in which a tumor, from its deep situation, almost always eludes discovery. Such cases are, however, discrimi-

nated by their presenting the same signs as a stricture of the œsophagus low down; indeed, they are very constantly combined with a narrowing of the tube, produced by the cancer spreading to it. Cancer at other parts of the organ occasions a perceptible tumor in about three-fourths of all the instances: its situation is, of course, not always the same. Where no tumor can be discerned, and particularly if, as may happen, portions of the stomach remain healthy and the digestive disturbances are slight, the existence of cancer may not reveal itself by any symptoms, and the case run a latent course.[1] In most cases without tumor we shall be rarely wrong in making the diagnosis of gastric cancer, if there be persistent stomach symptoms in a person of middle or of above middle age, whose digestion has been previously excellent, who has epigastric pain, is losing flesh and strength, is not improved by treatment, and shows an absence of hydrochloric acid in the trial meal.

A cancer of the anterior wall produces, as a rule, fulness, resistance, and percussion dulness in the epigastric region. A cancer involving the greater curvature gives rise to a swelling near the umbilicus, or to one extending towards either hypochondrium. The tumor formed by cancer of the pylorus is commonly felt plainly a little to the right of the median line, and one to two inches below the cartilages of the ribs. In women its position is apt to be even lower than this; and, indeed, in both sexes the situation of the indurated pylorus is very variable. It may be pushed down to near the umbilicus; nay, it has been discerned near the anterior superior spinous process of the ilium.[2] It is rarely found in the left hypochondrium, but not infrequently in the right. Then it may form adhesions to the liver, which viscus at times so completely covers the tumor as to render this impossible of detection.

The reason why the swelling, in not a few instances, shows itself much lower than the normal seat of the pylorus is obvious. During meal after meal the organ seeks to overcome the resistance offered by the narrowed pyloric orifice, and does so with great and increasing difficulty. The constantly repeated and long-continued struggle leads to hypertrophy of the muscular coat and to distention of the hollow viscus.

The tumor may or may not be movable,—generally is not; its surface may be either smooth or nodulated. It may be large and distinct, or small and requiring a careful examination to distinguish it

[1] See report of case under my care at the Pennsylvania Hospital, published in Amer. Journ. Med. Sci., vol. lii., 1866.

[2] See Lebert's cases in Traité pratique des Maladies cancereuses.

from the surrounding and more yielding textures. Percussing over it elicits a dull sound, usually mixed with a tympanitic note. The tumor is much more perceptible on some days than it is on others.

But is a swelling in the region of the stomach strictly pathognomonic of gastric cancer? No; not even when the swelling has been ascertained to belong to that viscus. At times the cicatrix marking a previous ulcer, or even the indurated and thickened margins of an existing ulcer, may be palpable through the abdominal walls and raise the question of a new growth. A mere fibroid thickening of the pylorus will occasion a tumor, and, moreover, produce symptoms which resemble so closely those of malignant disease at the orifice that I much doubt the possibility of distinguishing during life, with any certainty, between the two affections. Let us take this case, which I saw with Dr. Moss,[1] as an example.

A woman, aged forty, complained of pain at the pit of the stomach, and of a heavy sensation throughout the abdomen. For some months she had been suffering from indigestion, and had been losing flesh. She had a slight cough, with impaired resonance at the apices. The bowels were obstinately constipated, the tongue was smooth and red, the pulse feeble. She vomited shortly after meals, yet never anything but the ingesta. There was no pain on pressure over the pylorus; but a greater resistance to the finger than usual was detected. The further progress of the complaint was marked by incessant vomiting, only, however, after meals. Once, and once only, did it cease for several days; and then without apparent cause. As the case drew towards its fatal termination, the patient was much troubled with acid eructations, and had occasionally slight febrile attacks. The distress in the epigastrium increased. About three weeks before her death she was seized with lancinating pains under both patellæ; they were accompanied by pricking sensations and numbness in the legs, and an inability to walk. The pains gradually ceased, but the numbness and loss of motion increased. She died, utterly exhausted by the abdominal pains and the incessant vomiting, about three months after she began to reject her food. On post-mortem examination, tubercular deposits were found at the apices of the lungs. The abdominal viscera were healthy, except the stomach; and this, too, was healthy, save at its pyloric orifice, which was so narrowed that the tip of the little finger could hardly be forced into it. The mucous lining lay in folds, but on dissection was found to be perfectly normal. At the pylorus, but only there, the submucous and the muscular coat were uni-

[1] Published in full in the Proceedings of the Pathological Society of Philadelphia, vol. i.

formly thickened. Examined microscopically, they contained nothing but fibroid tissue, spindle-shaped fibre-cells, and very distinct organic muscular fibres.

Now, here is a case which was not cancer; yet it had the symptoms of cancer. It is true that the absence of blood and of glairy mucous in the matter vomited, and the indistinctness of the swelling, in spite of the great emaciation, were against the supposition of cancer of the pylorus. Still, no inference based on these data alone could be strictly trusted. The disease was combined with tubercular deposits in the lung. Nor is this the only example of the combination which has come under my notice. And when a tubercular state of the lung has been fairly made out, and there exist at the same time signs of pyloric obstruction, I should make a diagnosis that this is not of a cancerous nature, but consists simply of an increased development of the submucous coat, with probably subsequent hypertrophy of the muscular tunic.

The *fibroid thickening* may extend throughout the whole stomach, and there may be also hyperplasia of the muscular coat. Such cases differ from cancer by their long duration; the absence of hemorrhage, of the peculiar vomit of cancer, and of severe pain; and by the more uniform gastric swelling. The affection is sometimes observed in spirit-drinkers; it may be met with in children. Its discrimination from cancer is never a certainty. In a case reported by Cornell,[1] which was complicated with tuberculous peritonitis, loss of digestive power was indicated by unbroken starch grains in the vomit. The absorptive activity of the stomach tested by iodine was normal. Boas[2] states that in these non-malignant cases with pyloric stenosis, though there are fermentative processes, lactic acid is absent.

There are other diseases than those of the stomach which may occasion a tumor in its region and are thus liable to be mistaken for gastric cancer. Prominent among these are enlargement of the liver projecting into the epigastrium, tumors of the omentum, and diseases of the pancreas and of the kidney. Of course, the stomach symptoms proper are not so marked in these affections, and in some they may be wholly wanting; examination of the gastric contents and of the urine, and due regard to the history of the case, will show us the truth about many; and, after all, the best way of preventing ourselves from falling into error is to seek in any case of supposed gastric cancer for these other diseases, and to see if their chief symptoms are present.

[1] Montreal Medical Journal, Aug. 1892.

[2] Münchener Medicinische Wochenschrift, Oct. 1893.

Resting with this general statement, I shall not take up the differential diagnosis of all the many affections mentioned; especially as some are referred to when treating of partial abdominal enlargements and of cancer of the liver. But there are two which may be here specially looked at: one is omental cancer, the other kidney affection attended with marked swelling, such as occurs in hydronephrosis, pyonephrosis, abscess, hydatids, and morbid growths.

In *omental cancer* there is far less dyspepsia, hemorrhage and coffee-ground vomit are absent, the tumor appears to occupy chiefly the site of the greater curvature, the swelling is, or soon becomes, more generally diffuse, and hydrochloric acid and the digestive ferments are present in the gastric contents.

In the *kidney affections* referred to, the history is of great importance, and we include in this history the passage of renal calculi as bearing on some forms of kidney enlargement, especially abscess from impaction of stones; the limits of the mass, though this may project into the epigastrium, will scarcely be those of a gastric cancer. But the most certain safeguard against error is careful and repeated examination of the urine and of the gastric contents.

As regards the urine, the observations of Rommelaere[1] seem to show that its analysis may be of value in the diagnosis of the different forms of gastric disease. Thus, a cancerous ulceration of the stomach is attended with decrease of urea, and the chlorides are diminished. In simple gastric ulcer these are in normal quantity or in excess; so is the urea. In spreading gastric ulcer the chlorides are decreased, but there is a normal or increased amount of urea and urates.

In a certain number of cases, variously estimated between two and nine per cent., ulcer of the stomach exists first, and then *cancer supervenes*. This may take the form of a tumor, or the cancerous disease invade the ulcer, and no tumor occur. Such cases are mostly chronic, and present the history of preceding ulcer. The gastric juice generally retains its hyperacidity. There are often signs of a gastric neurosis; then loss of weight and cachexia are noticed, and want of response to treatment; all unlike pure gastric ulceration. A further significant sign is coffee-ground vomit. But the most conclusive would be furnished by the microscopic examination of particles of the morbid structure in washings of the stomach.

Dilatation of the Stomach.—This happens frequently in connection with obstruction of the pylorus, whether cancerous or fibroid,

[1] Journal de Médecine de Bruxelles, 1883; quoted in the Lancet, Sept. 1 and Oct. 27, 1883.

but it is also met with independently of this structural lesion. The latter form occurs from weakening of the muscular coats produced by malnutrition or impaired innervation, and has been noticed as an attendant upon anæmia or hysteria, or following fevers, or obstruction of the upper part of the bowel, or compression of the pylorus by an enormous gall-stone,[1] or, as Bamberger mentions, dislocation of the stomach by omental hernias. Edinger has proved that it may be associated with amyloid degeneration of the vessels or of the muscular coat of the stomach. The chief signs of a dilated stomach in either form are the rejection of food mostly in large quantities and retained for days; fermented and vomited matter containing often torulæ and sarcinæ; extension of the tympanitic note of the gastric region, detected by percussion, to much below the umbilicus; a splashing sound when the patient moves, particularly after drinking, and gurgling on sudden pressure; the low line of dulness occasioned by fluids in the distended organ, and the change of the dulness with the position of the patient; and slowly progressing emaciation. The character of the gastric secretion and that of the contents of the stomach after a trial meal vary with the nature of the causative condition. As a rule, there are increased acidity from the acids of decomposition and diminished absorptive and motor activity. The general nutrition suffers as assimilation is more and more interfered with. In doubtful cases the organ may be examined and its limits traced by distending it with ordinary air, or with carbon dioxide. Displacement of the right kidney has been observed in a number of cases.

The sounds of the heart heard over the dilated stomach often have a metallic ring, but, irrespective of this, peculiar gurgling sounds, systolic in rhythm, and evoked by the action of the heart, have been met with by Franck and other observers. Dilatation of the stomach may occasion serious nervous symptoms. I have known convulsions to occur, and tetany has been noticed.[2] The dilatation occasionally happens in an acute manner, and occurs in children[3] as well as in adults. As a rule, the muscular coat is not hypertrophied, but, in the cases in which an obstruction at the pylorus exists, this is frequent at first, ultimately giving place to atrophy.

To tell the atonic cases from those due to narrowing at the pylorus is generally not difficult; we can detect a hard swelling, or find the resistance with a stomach sound. In cancerous obstruction the gastric juice, as a rule, contains no hydrocloric acid, but we obtain lactic

[1] Minkowski, quoted by Ewald.

[2] Bulletins et Mémoires des Hôpitaux de Paris, t. xx., 1884.

[3] Archives Générales de Médecine, Aug. 1884.

acid. In other forms of stomach dilatation, particularly in the atonic form,[1] we find hydrochloric acid, as well as the acids of decomposition and fermentation, acetic acid, and butyric acid.

The stomach may be unduly large without giving rise to any symptoms. This condition of *megalogastria* is to be distinguished from gastric dilatation by the absence of the symptoms of the latter, as well as of derangement of secretion, absorption, and propulsion.

Enlargement of the stomach is to be distinguished from displacement of the organ,—*gastroptosis* or *Glénard's disease*. The condition is chiefly due to compression of the waist by the corset, or to relaxation of the ligamentous attachments of the stomach, produced by general debility and emaciation, or to weakness of the abdominal walls, such as follows pregnancies. Gastroptosis is often associated with a similar displacement of other abdominal viscera,—*splanchnoptosis* or *enteroptosis*. There are present, in addition to symptoms of digestive derangement and the obvious evidences of the dislocation of the viscera, which are best obtained by inflating the stomach with air or with carbon dioxide, manifestations of functional nervous disturbance. Among the first are impaired or perverted appetite, epigastric fulness and distention, eructation, acid taste and dryness of the mouth, burning or colicky pains at the pit of the stomach some hours after eating, diminished hydrochloric acid, pain in the back, and constipation alternating with seeming diarrhœa. The nervous symptoms include a feeling of weakness, general irritability, mental depression, headache or a sense of fulness in the head, vertigo, heaviness of the lower extremities, coldness of hands and feet, palpitation of the heart, heavy sleep, and frequently sacral pains. Emaciation takes place, with impoverishment of the blood, acne, and other changes in the skin, and falling out of the hair. In gastroptosis the lesser curvature of the stomach becomes evident after inflation, the pylorus is lowered, the organ is in a more vertical position.

A certain number of cases are associated with dilatation of the stomach. Gastroptosis is infinitely more common in women than in men,—ninety per cent. as compared with five, says Meinert,[2]—the smallest estimate places it at fifty per cent.

Dilatation of the stomach may be confounded with *dilatation of the large intestine*. But the gastric symptoms of the former malady are of great significance. Moreover, we may make use of the salol test in the discrimination. Salol is not acted upon by the acid gastric

[1] Germain Sée, Bulletin de l'Académie de Médecine, May, 1888.

[2] Centralblatt für innere Medicin, 1886, Nos. 12 and 13.

juice, but is changed into salicylic acid by the alkaline intestinal secretion. The salicylic acid manifests itself in the urine of healthy persons in from half an hour to an hour, as shown by the addition of a drop of tincture of chloride of iron into the urine giving it a violet color. In dilatation of the stomach salicylic acid does not appear for two or three hours after salol has been taken.

Hour-Glass Stomach.[1]—During digestion a constriction occurs near the middle of the stomach, almost entirely separating the cardiac from the pyloric half. This state may be a permanent one, and the *hour-glass* stomach produce decided symptoms. The hour-glass constriction may also be congenital, due to the contraction from a cancer or cicatrizing ulcer, or torsion from peritoneal adhesions. It is very rare under the age of twenty, and is vastly more common in women than in men. The history is frequently that of gastric ulceration with intense gastric pain and obstinate vomiting, often of food that has been for some time in the stomach; the more fluid parts of the ingesta are retained; there are apparent dysphagia, succussion splash in the lower part of the stomach remaining even after lavage, and a peculiar gurgling sound, described by Betz as *bruit de glouglou.*[2] Insufflation of the stomach, the gastrodiaphane, and the X-rays have also aided in the diagnosis, which is now assuming considerable interest, as hour-glass stomachs have been recognized and successfully operated on.[3]

SECTION II.

DISEASES OF THE INTESTINES AND OF THE PERITONEUM.

In considering the diseases of the intestines, we meet with symptoms the import of which we have examined in connection with affections of the stomach. We encounter nausea, vomiting, and impaired digestion. These may be sympathetic, dependent upon coexisting gastric disorder, or be the result of intestinal indigestion. In this the signs of indigestion are chiefly seen by the non-digestion and acid fermentation of starchy matter, and the incomplete action on fatty substances. Symptoms which in the study of intestinal affections we lay much stress on are pain and the character of the fecal discharges.

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[1] This has been investigated in an admirable paper on the shape and position of the stomach, by Bettmann, Philadelphia Monthly Medical Journal, March, 1899.

[2] Case of Jaworski, Wiener Medizinische Presse, No. 51, 1897.

[3] See reference to cases in Bettmann's paper quoted, and in Perret l'Estomac biloculaire, Thèse de Paris, 1896.

As regards the former, we draw the truest inferences from its kind rather than from its mere occurrence.

Alvine Discharges.—The fæces consist of about one-fourth solids and three-fourths water. *Dry*, hard stools depend upon an absorption of the fluid contents, as in constipation.

Watery stools are observed whenever a large quantity of the serum of the blood finds its way through the intestinal coats. They are met with after the administration of saline purgatives, in serous diarrhœa, and in cholera. Their hue varies: they may be almost colorless, or tinged with yellow. Sometimes, although very thin and watery, they are decidedly yellow; again they are rendered turbid by the dissemination of whitish flocculi, or cast-off epithelium, or by mucus. Whether they be yellow or colorless depends on the existence or non-existence in them of fecal matter and of bile. In a prognostic point of view, the most colorless evacuations are the most dangerous.

The presence of an excessive quantity of *mucus* renders the discharges less consistent than natural. The appearance they present is similar to that of the white of an egg; or the whitish masses of mucus surround the lumps of fæces, or are intermingled with the fluid alvine discharges.

Pus in large amount and unmixed with fæces is discharged only when an abscess has ruptured into some part of the intestine. Stools composed of fæces and pus are encountered in chronic inflammation and in ulceration of the bowels; and whitish, creamy streaks indicate the presence of the foreign substance. Yet the pus may be so intimately blended with the fæces, or with masses of mucus, as to require the microscope for its detection.

An excess of *bile* in the alvine discharges gives rise to evacuations of a yellowish brown or yellow hue. When the alimentary tube is highly acid, the resulting color is green. Both these kinds of stools are commonly called "bilious;" but the latter is less absolutely so than the former. A deficiency of bile manifests itself by clayey, sometimes even by almost white stools. Bile-pigment is not found in healthy stools. The stools may contain also concretions of biliary, pancreatic, or intestinal origin. Sometimes portions of neoplastic growths are appreciable to the naked eye. A curious and unusual form of concretion passing from the bowel is the so-called "intestinal sand." It resembles deposits of uric acid or urates, but does not respond to the tests for uric acid, as I have had occasion to note. It is supposed to be a substance intermediate between the ordinary bile-pigments and stercobilin.[1]

[1] Thomson and Ferguson, Journal of Pathology and Bacteriology, Feb. 1900.

Black stools result from eating certain articles of food, such as blackberries; from the action of medicines, as iron, bismuth, manganese; from a vitiated condition of the bile and intestinal secretions; or from the effusion of blood into the alimentary canal. At all events, when the hemorrhage proceeds from the stomach or the upper part of the canal, the stools have a black, tarry appearance; when from the lower section of the tube, pure blood is passed, or, if it be small in quantity, a blood-streaked mucus. Should any doubt exist as to whether the dark discharges be dependent upon the presence of blood, let them be diluted with water; they will assume a reddish tinge if this be the cause of the abnormal color. When blood pigment is present, it is in the form of hæmatin.

The *odor* of the evacuations is extremely offensive in fevers of a low type, and when the intestinal secretions are vitiated, or bile is absent. Acidity of the intestinal canal, as in the intestinal catarrh of children and of adults, or in rheumatism or gout, imparts to the stools a sour smell and an acid reaction. The reaction in health varies with the food; it is mostly alkaline.

In cases of constipation it may be important to notice the *shape* of the passages, because this may show whether an impediment has flattened or otherwise altered them. In fevers, as well as in affections of the intestinal mucous membrane, whether inflammatory or not, we often derive information from studying the form of the voided matter. Figured stools succeeding to fluid passages are always of favorable omen. We also note whether the stools contain masses of undigested matter and its kind.

Microscopical examinations of the fæces are not often made, but they may be of great service. They enable us, for instance, to recognize with certainty that the yellowish lumps contained in the evacuation, or the greasy film which collects upon its surface, consist of fat. The microscope, too, detects masses of muscular fibre, of elastic tissue, of starch-corpuscles, of fat, coagulated albumin, crystals of cholesterin, red corpuscles, leucocytes, and various fungoid growths, micro-organisms, and parasites. Among the animal parasites, besides various infusoria and worms,—the main variety of which will be discussed farther on with the parasites,—we find the *amœba coli*, now known to be the chief cause of tropical dysentery. It is one of the rhizopods, varying in size from 0.012 to 0.035 millimetre, and when active has a characteristic movement. This will be best seen if the stage of the microscope be kept warm.

The microscope exhibits, in the fecal discharges of all diseases in which the stools readily decompose, masses of crystals of the triple

phosphates; in acrid stools, yeast fungi; in typhoid fever, shreds of slough from the enteric ulcers, and bacilli; in tubercular ulceration of the bowel, tubercle bacilli; in cholera, comma bacilli; and under many varying conditions both in the fæces and in different organs, as well as in peritoneal exudates and in appendicitis, the *bacillus coli communis*. This is, as a rule, a sluggishly moving bacillus which grows readily on gelatin plates, the surface colonies being large and spherical and of a dull white. It is stained by aniline dyes, but is decolorized when treated by Gram's method. The main normal ingredient of fecal matter is mucin.[1] Phenol, indol, and scatol are common constituents. Peptone occurs only in disease.[2] One drawback to the use of *chemical* research for clinical purposes is the uncertain composition of the fæces, owing to the number of elements derived from the food. A large amount of starchy material shows deficiency of the diastatic ferments of the pancreatic juice in the salivary glands.

The study of the alvine discharges is of service not merely in intestinal complaints, but equally in the many maladies in which the alimentary tube sympathizes or becomes involved. Ocular inspection of the anal region may disclose the existence of hemorrhoids, fistulæ, fissures, or prolapse, and digital exploration of the rectum may yield information besides as to the presence or absence of ulceration, neoplasms, stricture, fecal accumulation, as well as to the tone of the sphincter and the sensibility of the mucous membrane, and also as to the condition of contiguous organs. The knowledge thus gained is supplemented or confirmed by ocular inspection with the aid of specula. The physical condition of the lower bowel may be investigated further by means of rectal insufflation of air or gas, or injection of water.

As a means of studying intestinal digestion, especially after test meals have passed from the stomach into the duodenum, the ingenious apparatus of Hemmeter[3] may be employed. The contents of the duodenum can be withdrawn and subjected to chemical and microscopical analysis. The activity af intestinal digestion and absorption may be estimated by the administration of two or three grains of iodoform in gelatin capsules hardened with formaldehyde; with gastric digestion, absorption, and motility normal, the saliva, tested with chloroform and nitric acid, will ordinarily yield the rose-red reaction of iodine in from four to six hours.[4]

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[1] Hoppe-Seyler, Handbuch. [2] Von Jaksch, Clinical Diagnosis, 1899.

[3] Johns Hopkins Hospital Medical Bulletin, April, 1895.

[4] Sahli, Deutsche Med. Woch., 1897, No. 1; Corresp.-bl. f. Schw. Aerzte, 1898, No. 10; Deutsches Archiv f. klin. Med., 61. B., 5. u. 6. H.; Lehrb. d. klin. Untersuchmeth., 2d ed., 1899.

But to review the uncomplicated intestinal diseases, grouping them as they may be recognized by pain and peculiarity in the fecal discharges, and describing with them the affections of the peritoneum.

Diseases attended with Paroxysms of Pain referred chiefly to the Middle or Lower Part of the Abdomen, and not associated with marked Tenderness or with Fever.

The type of these is colic.

Colic.—This is an intestinal pain, paroxysmal in its character, and usually combined with constipation, but unattended with febrile symptoms. The pain is of a severe griping or twisting kind, is commonly referred to the neighborhood of the umbilicus, and relieved by pressure. Sometimes there is soreness with the pain, and, indeed, a slight soreness not infrequently remains after the paroxysm has passed off. While the pain lasts, the countenance wears an anxious, frightened expression; the skin is cold; the pulse is depressed. Occasionally there is vomiting, and in severe cases the abdominal walls are tense or raised in hard knots by the spasmodic contraction of the muscles. An attack may last only a few minutes, or for several hours.

Some persons are very liable to attacks of colic. Those who suffer from indigestion, or are enfeebled by exhausting maladies, are predisposed to them; so also are hysterical, gouty, and rheumatic individuals. As to the exciting causes, they are various; and somewhat according to its different causes, colic presents different forms. Let us indicate the more prominent.

Colic, simple and unconnected with a disease of the bowel.—In these cases, generally called spasmodic colic, the paroxysmal pain may be of diverse origin. It may be the result of direct excitation of the peripheral intestinal nerves by the presence of irritating substances in the canal, such as indigestible food, cold or acid drinks, hardened fæces, gases, morbid secretions, ptomaines, worms, medicines, or poisons. It may proceed from an irritation of the central nervous system reflected to the intestinal nerves. It may be sympathetic, and produced by a morbid state of the adjacent abdominal viscera.

1. Colic owing to food difficult of digestion is very common, especially at the time of year when fruit is beginning to ripen. It may be caused by food taken in quantities greater than the digestive organs can assimilate. Hence it is frequent in children at the breast who are overnourished, and in persons in delicate health with enfeebled digestive powers. The form of colic under discussion is often attended with vomiting and diarrhœa; it may be of only a few hours' duration, or it may last for several days.

Colic arising from distention of the intestines with flatus, or "flatulent colic," is the result of the decomposition of food in the alimentary canal; sometimes, however, the gases are extricated from morbid secretions, or are exhaled directly from the blood-vessels. The abdomen is distended, and the flatus is from time to time discharged, with evident relief. Hysterical persons are subject to this form of colic.

Colic from the presence of morbid secretions in the intestinal canal is not so often encountered as that from indigestible food or retained fecal matters. Yet it is occasionally met with in cases of diarrhœa attended with a disordered state of the intestinal functions; in the so-termed bilious colic the intestinal pain is produced by the irritating character of the bile.

This bilious colic is often preceded by nausea, loss of appetite, and a coated tongue. The paroxysms of pain frequently go hand in hand with vomiting,—first of the contents of the stomach, then of bile. They are in general accompanied or soon followed by a yellowish tinge of the conjunctiva, by tenderness in the region of the liver, and by a desire to go to stool. The bowels are, however, apt to be obstinately constipated. Bilious colic is common in malarious districts; it occurs especially during the summer and autumnal months, and frequently follows exposure. It sometimes begins with a chill, and, unlike the other forms of colic, it has as companions febrile excitement, and a full, frequent pulse. Malarial colic may occur in an epidemic form.[1]

2. In the second class of cases belong colic arising from fright from anger, that happening in nervous females and hypochondriac males, perhaps that proceeding from sudden exposure to cold, the form which is sometimes seen coexisting with neuralgic pains in other parts of the body; in short, all those cases which are spoken of as nervous colic. The attack is sudden, and not commonly of long duration; but it is apt to be repeated.

The "metallic colics" are further instances of colic produced through agents which act primarily on the nervous system. This is certainly true of lead colic. *Copper colic* exhibits paroxysms of severe pain like those caused by the poisonous influence of lead; but it is attended with nausea, vomiting, diarrhœa, tenesmus, an abdomen distended and tender to the touch,—in other words, it is rather an inflammation of the intestine with colicky pain than uncomplicated colic. The distinguishing marks of *lead colic* are the bluish-gray line along the gums; the contracted abdomen; the obstinate constipation;

[1] American Journal of the Medical Sciences, April, 1872.

the great relief to the pain usually afforded by pressure; the duration of the pain; its marked and agonizing exacerbations; and the history of the case. The signs of the lead poisoning also manifest themselves in other parts of the body.

3. Affections of various organs may give rise to colic, by sympathy, and generally through irritation reflected through the nervous system. Thus, colic is a not uncommon attendant on morbid states of the kidneys, liver, bladder, testicles, uterus, or ovaries, and on disordered menstruation. Yet we must not forget that the pain, although spoken of as colic, is often not strictly intestinal, but is merely a pain radiating from the affected organs themselves. Again, how far it is due to neuritis is a matter to be taken into account.

Colic arising in consequence of some abnormal state of the bowel.—But colic may have only the significance of a symptom, and be combined with an altered structure or a changed position of the intestine. We meet, indeed, with colicky pains in dysentery; enteritis; hernia; ulceration; intussusception; strangulation; twisting; strictures; distention,—in fact, in the most various morbid states of the intestine. And colic as a symptom can be discriminated, as far as the pain is concerned, from colic as an idiopathic disorder, only by the history and the concomitant phenomena of the case. In several of the maladies cited the more transitory nature of the pain,—or gripings,—in others the presence of fever and of tenderness, serve as guides in diagnosis. Fever and soreness to the touch are also met with in that form of inflammation of the bowel which happens after exposure, or after the retrocession of rheumatism from some external part, and which is commonly known as rheumatic or inflammatory colic.

The disorders with which uncomplicated colic, or that which is held to be purely spasmodic, may be confounded, are:

GASTRALGIA;
APPENDICITIS;
PERFORATION OF THE INTESTINE;
STRANGULATED HERNIA;
PASSAGE OF GALL-STONES;
NEPHRALGIA;
SPASM OF THE BLADDER;
UTERINE COLIC;
NEURALGIA OF THE DORSAL AND LUMBAR NERVES;
ABDOMINAL ANEURISM AND TUMORS; DISEASES OF THE SPINE;
ENTERITIS AND PERITONITIS.

Gastralgia.—In gastralgia the pain is seated in the epigastric region; whereas in colic, or enteralgia, the pain is near the umbilicus,

or rapidly shifts its position from this point to different parts of the abdomen, and is often connected with a spasmodic contraction of the abdominal muscles. Again, the history in cases of gastralgia; the fact that the attacks happen most frequently after meals; their association with signs of a disordered stomach,—indicate the organ in which the pain arises. And much the same general signs, in addition to the marked constipation and the visible movements, enable us to distinguish those instances of peristaltic disturbance of the stomach to which Kussmaul[1] has called attention, and in which the drawing pain is apt to be referred to the intestine: indeed, the peristaltic disorder may spread to it.

Appendicitis.—The sudden and sharp pain of appendicitis, occurring in paroxysms and often following acute digestive disorders, is very apt to be mistaken for colic. But the seat of the pain, which is generally in the right iliac fossa and which becomes associated with tenderness and with fever, tells us the condition we are dealing with.

Perforation of the Intestine.—When paroxysms of pain have their origin in perforation of the intestine, the extreme prostration and collapse show that they are not produced by a harmless disorder like colic. Further, the abdominal distress is in most cases preceded by symptoms of a diseased state of the stomach or the intestines, of appendicitis or of typhoid fever; and if the patient live sufficiently long after the accident, the pain is followed by distention of the abdomen and extreme tenderness,—in fact, by the signs of peritonitis. However, the differential diagnosis is occasionally very difficult. A valuable sign of perforation and of air in the peritoneum is the obliteration of the dulness on percussion over the hepatic region, pointed out by Alonzo Clark.

Strangulated Hernia.—All mechanical obstructions of the intestine will lead to paroxysms of intestinal pain. They are met with in cases of intussusception and of ileus; they are also frequent in cases of strangulated hernia. In all, the obstinate constipation should arouse suspicion regarding the true nature of the complaint. To detect a hernia a local examination is required; and, therefore, a careful search at the usual seats of this affection ought to be made in every instance of severe or protracted colic. Lives have been lost in consequence of the neglect of this simple precaution against disastrous error.

Passage of Gall-Stones.—The passage of a gall-stone is generally attended with paroxysms of intense pain which are readily mistaken for colic. There is, as a rule, the same absence of fever and of ten-

[1] Sammlung klinischer Vorträge, No. 181, June, 1880.

derness; yet fever of short duration does happen. Pressure is often resorted to in order to mitigate the suffering, and thus the resemblance to colic is heightened. The points of distinction from colic are, the position of the pain in the epigastric region; the severe nausea and vomiting attending the attack; the jaundice; and the voiding of gall-stones with the stools. The latter sign, though a positive one, assists less in the discrimination of the disorder than would appear at first sight; partly because it does not serve as a means of indicating the nature of the affection until its close, partly because the calculus often escapes detection in the fæces. The best way to find it is to pass the evacuations through a sieve; this is more certain than covering the discharge with water. The stone may not come from the bowels for some days after the attack of colic. Its passage gives rise to symptoms like those of bilious colic. The repetition of the attack is always a strong reason for suspecting it to be owing to a discharge of a calculus from the gall-bladder; and so are severe retching and vomiting, sudden supervention of jaundice, and localized epigastric pain. *Hepatic neuralgia*, if there be such a disease, cannot be discriminated from gall-stone colic, except by its recurrence after certain intervals, the alternations with other affections of the nervous system, and the slightest touching of the part inducing at times the acute pains.[1]

Sometimes gall-stones are closely simulated by impacted fæces, the pressure of which occasions colicky pains, and even jaundice. A dose of oil brings away the hardened fæces. The swelling in the right side may be sometimes readily detected. Among the rarer symptoms attending or following the passage of gall-stones, temporary dilatation of the heart and tricuspid regurgitation have been noticed.[2]

Where the gall-stones are large and have become impacted in their course towards the intestine, they give rise to inflammation which may lead to ulceration and to the discharge of the concretion—generally then very large—into the intestine or stomach. Subsequently an obliteration of the duct may happen; or the inflammation and ulceration of the duct may result in perforation into the peritoneum. In some cases the gall-stones are voided through the abdominal walls, in consequence of their having caused inflammation of the gall-bladder and subsequent adhesions to the abdominal parietes. The fistulous passages discharge pus and bile, and occasionally fresh stones: they may last for years, but in time they generally heal.

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[1] See the cases of Budd, on Diseases of the Liver; of Andral, Clinique Médicale, tome ii.; and of Frerichs, Diseases of the Liver.

[2] Potain, quoted by Sée, Maladies du Cœur, Paris, 1883.

Nephralgia.—Paroxysms of pain with intervals of comparative ease and unassociated with fever occur in nephralgia, and are often mistaken for colic. Now, kidney pain is generally, although not invariably, caused by the passage of a calculus through the ureter. Its symptoms, besides the pain, are numbness of the thigh, nausea and vomiting, a constant desire to make water, and aching and drawing up of the testicle. The patient, as in colic, is restless, and seeks relief by frequently changing his position. The pain comes on suddenly, and is excruciating. It is felt in the loins, usually on one side, and shoots along the track of the ureter to the hip and thigh, or extends to the umbilicus; it is often associated with tenderness in the course of the ureter. Occasionally it is almost exclusively felt at the hip. When the stone reaches the bladder, the pain ceases as abruptly as it began; though sometimes there is still discomfort produced by the stone interfering with the act of micturition. During the attack the urine is passed in small quantities at a time. It is high-colored; sometimes it contains a little blood. If it be collected, after all pain has disappeared, and be carefully examined, a small, hard body or a sandy deposit is generally detected, and reveals the cause of the past anguish. It is from the presence of the sandy deposit that the complaint has received popularly the name of a fit of "the gravel."

The seat of the pain is a chief distinction from intestinal colic; yet in neither complaint is the seat entirely characteristic. It is not always strictly umbilical in colic; it is not always exactly in the region of the ureter or kidney in nephralgia. Of more importance is the state of the urinary functions, which are comparatively undisturbed in colic. Again, the numbness of the thigh and the retraction of the testicle are valuable diagnostic marks; they would be absolutely decisive were they constantly present in nephralgia.

Spasm of the Bladder.—The bladder is sometimes the site of paroxysms of violent pain, supposed to attend upon a spasm of the viscus. There is an intense desire to urinate, which the passing of water does not allay. The pain is accompanied by a sense of constriction at or near the pelvis, and sometimes by tenesmus, and may extend to the kidneys, to the thighs, and to the sacrum; or the irritation may be communicated to the penis, and cause erections. If the sphincters be involved the urine cannot be voided. The bladder distends; there is intense anxiety, with restlessness; the pulse is feeble; the skin is cold, and covered with clammy perspiration.

A spasm of the bladder may be caused by the presence of a stone or of irritating urine. It is also encountered in gout and hysteria, and as the result of stimulating diuretics. Violent fright, too, may occasion

it. It sometimes proceeds from a disorder of adjacent structures, as of the rectum or of the uterus. Now and then it is associated with inflammation or suppuration of the kidney, and the vesical pain is so intense that it withdraws attention from the organ most affected. To distinguish it from colic is not difficult; the location of the pain and the disturbed condition of the urinary functions serve as guides. It resembles nephralgia more closely.

Uterine Colic.—The painful sensations experienced by some women at their menstrual periods may come on in paroxysms similar to those of colic. In truth, the pain is often spoken of as uterine colic, and at times continues for many days, persisting during the menstrual period, or even longer. In some of these cases the complaint is localized in the uterus; in others, in the ovaries, which are then tender to the touch.

Now, with reference to the disorder first mentioned, or ordinary dysmenorrhœa, it may be easily discriminated from colic by its occurrence with the setting in of the menstrual flow; by the pain remitting rather than intermitting; by the seat of the pain in the pelvis, or the lower part of the abdomen; by its not uncommon association with nausea and vomiting; and by the fact that all the signs of disordered menstruation have happened previously at the periods.

Where the ovaries are much congested or inflamed, whether or not the affection exist in connection with dysmenorrhœa, or occur in consequence of other causes, among which gonorrhœa may be one, the pain, tenderness, and swelling in the hypogastric region; the numbness and flexed position of one or both thighs; the febrile irritation, and the hysterical symptoms; the retention of the urine; the violence of the paroxysms of pain, and the duration of the malady,—form a group of phenomena very dissimilar to those of ordinary cases of colic.

Neuralgia of the Dorsal and Lumbar Nerves; Abdominal Neuralgia.—The dorsal and lumbar nerves are subject to neuralgic affections, which exhibit, like colic, paroxysms of pain unaccompanied by fever. But Valleix has taught us to look for spots painful to the touch in the course of the aching nerves, and has shown that the disturbance of the nerves supplying the abdominal parietes manifests itself on one side of the body only, whereas an irritation of the intestinal nerves obeys no such law.

In neuralgia of the lumbar nerves, or *lumbo-abdominal neuralgia*, the pain is commonly felt in the hypogastric region, a little to one side of the median line. In this situation, too, there is localized soreness on pressure; the other tender spots are, generally, one a little to the outside of the first or second lumbar vertebra, and one immediately

above the middle of the crest of the ilium. In women, who are by far the greatest sufferers from the disease, there is sometimes also a painful place about the middle of the Fallopian tube, or on the neck of the uterus; in men, a point on the scrotum here and there is found sore to the touch. These spots of tenderness serve as characteristic signs; and they enable us to separate neuralgia not only from colic, but also from lumbago, and from rheumatism of the abdominal walls.

Besides these forms of neuralgia we find other kinds of abdominal neuralgia, which may be mistaken for colic. They are attacks of pain of great severity, affecting especially the mesenteric plexus or the solar plexus, and attended with a sense of faintness and annihilation. The disorder is often excited by exertion, is associated with debility, and relieved by an antineuralgic treatment. In some cases it is of malarial origin; and in every instance we must lay stress on the frequent recurrence of the pain and on the history to enable us to discriminate between the neuralgic complaint and colic. The distinction from gastralgia can be made only by the more marked gastric symptoms, and by the absence of marked prostration and sense of fainting in this malady.[1]

Angioneurotic Œdema.—The local œdematous swellings of passing character met with in this disease are generally associated with attacks of severe colic, nausea, and vomiting. In recurring purpura the same occurrences are met with.

Abdominal Aneurism and Tumors; Diseases of the Spine.—In all of these we may find violent pain of a paroxysmal kind referred to various portions of the abdomen, and unaccompanied by fever. We judge that the pain is not colic by its frequent repetition; by its want of association with intestinal or gastric disturbance; by its being, although liable to exacerbations, so steadily present at some part either of the spine or of the abdomen; and by the attending symptoms and signs occasioned by an abdominal tumor, or by a disease of the lower dorsal or of the lumbar vertebræ.

Enteritis and Peritonitis.—Inflammations of the intestines and of the peritoneum also give rise to severe abdominal pain. But it is more constant, and is linked to great tenderness, and, in acute cases, to symptoms of high febrile excitement. Thus enteritis and peritonitis belong to a different group of diseases,—a group of inflammatory affections, which I shall now describe.

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[1] A number of cases of abdominal neuralgia are reported by Handfield Jones in his Treatise on Functional Nervous Diseases; and by Porcher in American Journal of the Medical Sciences, July, 1869.

Diseases attended with Pain and Marked Tenderness in the Umbilical Region or diffused over the Abdomen.

Acute Enteritis.—Enteritis means, by common consent, inflammation of the small intestine, especially of the portion that lies between the duodenum and the colon. The morbid process may extend to the colon; if, however, it involve a large portion of the latter, it is colitis or dysentery. There are two forms of enteritis; one in which the mucous membrane of the bowel is alone affected,—muco-enteritis or intestinal catarrh. In the second, more than the mucous tunic is implicated; there is also inflammation of the submucous and muscular coats, or even of the serous investment of the bowel. To this variety of the complaint the term enteritis is by several writers restricted; and it is to this rare form of the malady, a phlegmonous enteritis, occurring acutely, that the description about to be given more particularly applies.

The symptoms of an acute attack of enteritis are those of colic, attended with fever and tenderness. The disorder may begin with the symptoms of colic, or it may set in with chill and fever. When the disease is fully established the fever runs high; the pulse, tense and full at the onset, becomes small and wiry. There are nausea and vomiting, and sometimes distressing fits of retching. The tongue is covered with a white coat, or is red and dry. The bowels are constipated; sometimes there is diarrhœa, or constipation alternating with diarrhœa. The stools may contain a small quantity of blood; they rarely contain pus. The appetite is lost, the thirst great. The pain, as in colic, is paroxysmal. It begins near the umbilicus, and thence may shift to various parts of the abdomen, but not to the epigastrium; it does not cease as in colic, but rather exacerbates, and then changes to a dull feeling of distress. It is greatly increased by pressure, and the patient seeks relief, as in peritonitis, by lying on his back with his thighs flexed, so as to relax the abdominal muscles. Towards the right of the umbilicus it is not uncommon to find a marked pulsation, from throbbing of the abdominal aorta or of its large branches,—a sign to which Stokes[1] directed attention. This pulsation may be very annoying. In looking over the notes of my cases on which the description of the symptoms of enteritis just given is based, I find one in which neither the thirst, nor the pain, nor the nausea and vomiting occasioned as much distress as the violent throbbing in the abdomen.

[1] Article "Enteritis," in Cyclopædia of Practical Medicine.

In the instances of the malady that advance to a fatal termination, the pulse becomes quick and irregular; hiccough appears; the abdomen swells; the features are haggard; and the patient's strength becomes gradually exhausted. The worst and most hopeless cases of the disease are those dependent on mechanical obstruction of the bowel, whether it proceed from organized bands in which a loop of intestine is caught, or from invagination, or from accumulation of hardened fæces, or from a hernial strangulation. The disease may lead to purulent infiltration of the submucous tissue and to abscess-formation.

Among the symptoms of enteritis, pain is one of the most important. It is never absent, save in rare instances in which the inflammation is very intense at the onset.[1] Still more important is the great tenderness. This enables us to say that the case, in spite of the colicky pains, is not colic. It warns us not to administer strong cathartics to overcome the constipation that appears in consequence of the severe inflammation.

The disease in its violent form just described bears a close resemblance to peritonitis: we shall presently see what are its distinguishing marks. But there is, as above stated, another variety of the disease, a mild variety, or *muco-enteritis*, in which the disturbance is limited to the mucous membrane. The main features of this intestinal catarrh are the same, but they stand out in less bold relief. There are griping pains, a slight soreness to the touch, general uneasiness, loss of appetite, thirst, nausea, and sometimes vomiting. But we find only slight fever; and the febrile excitement remits in the morning. Diarrhœa is present, and the stools are sometimes very offensive. This form of the disease may terminate, as the severer inflammation generally does, in less than a week; yet it may persist for several weeks, and thus gradually lapse into a chronic complaint. It is common in children, especially during dentition. It is also observed when irritating food or secretions occupy the alimentary canal for any length of time, or after exposure to cold and damp, particularly when the skin is perspiring freely, and as an attendant upon the exanthemata. It resembles typhoid fever, Indeed, it is sometimes difficult, especially in children, or in the intestinal catarrh of catarrhal fever, to know whether we are dealing with a case of simple intestinal catarrh or with the intestinal symptoms of enteric fever. The state of the cerebral functions, the pain and gurgling in the iliac fossa, and the high temperature in the latter malady, may

[1] Andral, Pathologie interne, tome i. p. 47.

clear up the doubt; yet in some cases nothing but the eruption and the results of the Widal test will do so.

The symptoms just described belong to catarrh of the ileum, or of the ileum and the ascending colon. In catarrhal inflammation of the duodenum there is often constipation in place of diarrhœa. Pain between two and three hours after the taking of food, loss of appetite, coated tongue, fetid breath, marked digestive disorder, flatulency, and jaundice are prominent among the symptoms. The pain is apt to come on in paroxysms like gastralgia, although referred somewhat lower than the stomach; these seizures last several hours, and slowly subside. We frequently find a certain amount of soreness developed by deep pressure in the right hypochondrium and the upper part of the umbilical region. There is weakness, with much despondency, and slight elevation of temperature. An acute attack lasts two or three weeks. In the chronic form the duration may be as many months.

Another affection which is liable to be mistaken both for enteritis and for typhoid fever has been described by Klob.[1] The chief symptoms are violent pains in the hypogastric region, with vomiting, thready, frequent pulse, high temperature, and the rapid supervention of somnolence and coma. In some instances hemorrhages happen. Hemorrhagic erosions are found in the stomach, and bloody infiltrations in the jejunum; the parenchyma of the mesenteric glands, their lymphatics, and the thoracic duct are infiltrated with blood; the spleen is enlarged. The disorder shows then a striking hemorrhagic tendency, and is supposed to be a blood-affection similar to pseudo-leukæmia.

A *croupous* or *diphtheritic* enteritis is not seen save as a secondary process, if we except the instances in which it follows poisoning by mercury, by arsenic, or by lead. It is more generally encountered as a secondary affection in some infectious diseases, as in pneumonia, pyæmia, typhoid fever; or in cancer, Bright's disease, or cirrhosis of the liver. Its symptoms may be latent, but generally there are diarrhœa and pain without tenesmus.

Acute Peritonitis.—As in acute enteritis, so in acute peritonitis, pain and tenderness are the most significant symptoms. To these are joined fever, distention of the abdomen, and, frequently, cold sweats, nausea, vomiting, and obstinate constipation. The disease begins with chilly sensations or protracted rigor. To these succeed fever, and abdominal pain and distention. The fever runs high at the onset; it exhibits a dry, burning skin, an axillary temperature of 103°

[1] Wien. Med. Zeitung, quoted in Lond. Med. Record, Feb. 1875.

and upward, a pulse frequent, but, as in acute inflammations of the mucous and serous membranes below the diaphragm, small and wiry. However, both the character of the pulse and that of the skin change as the malady progresses. The pulse is less tense and more developed as the inflammation subsides, or feeble and flickering if the disorder proceed towards a fatal termination. The skin is frequently covered with cold sweats. The temperature is irregular, and may sink below the normal. The features are sharpened and wear the look of death, even in cases which ultimately recover.

The pain is constant and severe. It may exacerbate, but it never intermits. At first the pain is confined to a particular point; but as the inflammation extends, so it extends over the whole abdomen. It is increased by the slightest pressure, be that pressure exerted by the hand or by movements of any kind. To obviate the pressure, the patient lies on his back with his thighs flexed, and, however tired of retaining the same position, he does not change it. The descent of the diaphragm augments the pain: instinctively, therefore, he refrains from drawing long breaths, and his respiration is short and frequent and purely thoracic.

The abdominal distention is in part owing to meteorism, in part to the liquid effused into the peritoneum. Percussion tells us in individual cases how far each factor acts as a cause of the enlargement by the tympanitic or the dull sound elicited. Palpation, too, reveals the presence of liquid. Yet percussion or palpation ought to be employed only with the greatest care, on account of the pain they occasion. The fluid does not gravitate as invariably as in ascites to the lower portion of the belly. It is often caught in sacs formed by the membrane adhering in spots; and thus circumscribed dulness may be found at one or several parts of the abdomen. Sometimes the roughening of the membrane gives rise to a distinct friction sound.

Independently of the abdominal pain and swelling, we meet, in acute peritonitis, with constipation, nausea and vomiting, headache, a suppression of the urinary discharge, and in rare instances with priapism; of these symptoms, constipation is the most constant. It is caused by the paralyzed state of the intestine, to portions of which the inflammation may spread; or by the lymph gluing together the coils of the bowels.

Death in acute peritonitis is commonly preceded by enormous tumefaction of the belly, cold sweats, a pinched countenance, and a rapid, flickering pulse. When recovery takes place—unfortunately a rarer issue of the malady than its fatal termination—it is very slow and gradual: and often morbid conditions remain which prolong

greatly the patient's illness, and may lead in themselves to a disastrous result. It is, therefore, impossible to foretell the duration either of the acute disease or of its consequences.

Acute peritonitis arises idiopathically from exposure to cold and wet only very occasionally; much oftener in consequence of injuries to the abdomen, such as blows, stabs, or kicks; or from perforation or laceration of some of the abdominal organs, such as perforative ulcer of the stomach, intestine, appendix, or gall-bladder, and discharge of their contents into the peritoneal cavity, or from a ruptured tubal pregnancy. Uterine injections passing into the peritoneal cavity may cause peritonitis. It also results from rheumatism, or from a poisoned state of the blood, or from acute tuberculosis, or from Bright's disease. It sometimes originates from an inflammation of the abdominal viscera, especially of the spleen, intestines, or uterus and its appendages, spreading to their serous covering. Again, other morbid states of the abdominal organs, such as cysts of the ovaries, intestinal intussusception, or strangulated hernia, may compress or irritate the membrane, and lead to inflammatory action.

Perforative peritonitis is characterized by its sudden development. Most frequently perforation of the stomach or intestine or appendix lies at the bottom of the mischief, and many of the cases are met with in typhoid fever, or in disease of the gall-bladder, or in salpingitis. Whatever its cause, the perforation is attended with severe pain, sometimes with a chill, and is immediately followed by collapse; tenderness and distention of the abdomen, and shallow breathing, vomiting, and rise of temperature soon make their appearance. The swelling of the abdomen is great, and the gas in the peritoneal cavity occasions tympanitic resonance over the liver and spleen, obliterating the normal dulness of these organs. In very rare instances the contents of the alimentary canal may be discharged into the sac without giving rise to inflammation.[1]

The peritonitis of childbed fever, or *puerperal peritonitis*, is in its symptoms, so far as the peritoneal inflammation is concerned, not different from those of any other kind of peritonitis, except that diarrhœa, instead of constipation, is often present. The disease is generally ushered in by chills. The temperature rises speedily to a considerable height, to 104° or 105°, and continues high with irregular remissions. It is in the region of the uterus or the uterine appen-

[1] Cases reported by Bardeleben and Siebert, quoted in Henoch's Clinic of Abdominal Diseases. I have met with several instances of the kind in typhoid fever.

dages that pain and tenderness are first felt. But, independently of the symptoms of the local disorder, there are evidences of a septicæmia; we find delirium, black vomit, exudation into the pericardium and pleura. Fortunately, the diagnosis is one we are now less and less often called upon to consider, for antisepsis has almost put a stop to the disease.

Partial or local peritonitis is almost invariably owing to a preexisting morbid condition of some abdominal viscus. Sometimes the circumscribed inflammation is protective rather than calculated to work mischief. It arrests a destructive perforation of the membrane, or it limits the matter discharged to a certain spot; it may at least do so for a time, for general peritonitis is very apt ultimately to follow.

Partial peritonitis often pursues a subacute rather than an acute course. It may end in adhesions or lapse into a chronic state. Its symptoms are much the same as those of a more general inflammation,—the same fever and constipation, the same pain and tenderness. The fever does not, however, run so high, and the pain and the great tenderness are much more localized. The abdomen, also, is not so swollen or so tympanitic. But perhaps even more frequently than in general peritonitis are found accurately limited spots of dulness on percussion corresponding to circumscribed exudates or collections of pus in the peritoneal cavity.

Partial peritonitis is more liable than the general disease to be confounded with other disorders. Yet error can hardly arise if we bear in mind that it is precisely with the morbid states of the viscera which lie below the peritoneum that the circumscribed inflammation of the serous membrane is usually connected, and that local peritonitis, therefore, frequently attends the very disorders from which we seek to distinguish it. Let us, however, examine into some of the complaints with which peritonitis, whether local or general, may be confounded. They are—leaving for consideration elsewhere obstruction of the bowel, appendicitis, and perityphlitis—

Acute Gastritis;
Acute Enteritis;
Acute Pancreatitis;
Metritis;
Cystitis and Distention of the Bladder;
Rheumatism of the Abdominal Walls;
Abdominal Hysteria;
Colic.

Acute Gastritis.—Acute inflammation of the stomach can scarcely be mistaken for inflammation of the peritoneum, provided attention

be paid to the history of the case and to the seat of the pain. The former disorder begins with vomiting, and this continues a prominent symptom; whereas vomiting is not so constant, nor does it occur so early, in peritonitis. The pain and tenderness are limited to the region of the stomach in gastritis; they are diffused in peritonitis. They may, it is true, be localized when the peritonitis is partial. But acute inflammation of the gastric peritoneum is hardly encountered, save as an attendant on severe inflammation of the stomach, or on destruction of its coats,—the form of gastritis which results from irritant poisons.

Acute Enteritis.—Enteritis differs from general peritonitis by the less extended tenderness; by the seat of the pain near the umbilicus, and its more paroxysmal character; by the comparative absence of tympanites and abdominal tumefaction; and by the greater prominence of nausea and vomiting. Yet it cannot be distinguished with certainty from the partial form of acute peritonitis, to which, in truth, some of its symptoms are clearly owing.

Acute Pancreatitis.—This is a cause of peritonitis easily overlooked. The pancreatic inflammation mostly arises in consequence of the extension of a gastro-duodenal inflammation along the pancreatic duct; or it may follow hemorrhage into the pancreas. In the former case we find sudden pain, deep-seated, constant or paroxysmal; tenderness; and tympany in the epigastrium in the region of the pancreas, with nausea and vomiting. This is gradually followed by peritonitis at the same place, and by a low fever. Constipation is frequent, and, with the other symptoms, has led to the diagnosis of acute intestinal obstruction and to laparotomy. The symptoms of acute panrceatitis may be also produced by extensive fat necrosis of the pancreas. In *hemorrhagic pancreatitis* the malady runs a rapid course. The disease occurs in persons over thirty years of age. The attack begins with violent pain in the upper part of the abdomen; nausea, vomiting, and abdominal swelling soon follow, and delirium and signs of collapse appear. There is usually constipation. The temperature, as we know from Fitz's[1] comprehensive study, may remain normal. The disease is most likely to be confounded with acute perforative peritonitis. It usually proves fatal in from two to four days. The hemorrhage may lead to gangrene; in either case the signs of peritonitis are marked. Hemorrhage may occasion sudden death.[2] *Suppurative pancreatitis* has much the same symp-

[1] Middleton-Goldsmith Lecture for 1889.

[2] Draper, Transactions of the Association of American Physicians, 1886.

toms; but it does not run so acute a course,—is, indeed, often chronic; there is apt to be irregular fever. In a case that I saw with Dr. Hulshizer, the pain was severe but paroxysmal, repeated chills occurred, there was sugar in the urine, and decided polynuclear leucocytosis.[1]

Metritis.—In this the pain on pressure is confined to the uterus and its annexes, and there is little or no tympanites. In puerperal peritonitis with metritis, the signs of inflammation of the serous membrane mask those of inflammation of the womb.

Cystitis and Distention of the Bladder.—Both inflammation and distention of the bladder are occasionally mistaken for general acute peritonitis. An acute inflammation of the bladder gives rise to frequent calls to pass urine: yet the act is performed with great difficulty, and in severe cases may become impossible; the bladder distends; a sense of uneasiness is felt in the perineum; the region above the pubes becomes tender, and sounds dull on percussion; there is great restlessness, fever; at times vomiting and hiccough supervene. Such cases resemble those of peritonitis with suppression of the urinary discharge and with strangury. But the urine voided in peritonitis is simply high-colored, like that of any febrile state. In cystitis it contains large quantities of mucus and pus, and often blood and crystals of phosphates. Again, the abdominal tenderness is localized, and is frequently accompanied by a smarting in the course of the urethra. Neither of these signs is encountered in peritoneal inflammation, and, as a rule, the temperature in this is higher. The urinary disturbance which not infrequently takes place in the latter disorder is attributable to inflammation of the peritoneum covering the bladder.

An over-distention of the bladder, not the result of inflammation of its coats, may produce a local tenderness spread over a considerable portion of the lower part of the abdomen. But the outline of the dulness, which is the same as that of the tenderness, the fact that the patient has generally not passed urine in any quantity for a considerable time, the almost normal temperature, and the sudden cessation of the supposed peritonitis on passing a catheter, show the true nature of the malady.[2]

Inflammation and Abscess in the Abdominal Muscles.—When the abdominal walls become inflamed, symptoms are occasioned that are

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[1] Philadelphia Medical Journal, June 11, 1898.

[2] A case of this kind, occurring after delivery, is given by Lever, Guy's Hospital Reports, 2d Series, vol. viii. p. 41.

not always easily distinguished from those of acute peritonitis. The disease is attended with some fever, with pain increased by movement, by the act of coughing, and by pressure, and sometimes with excessive tenderness. The seat of the inflammation is generally the rectus muscle and the surrounding cellular tissue. The parts on one side of the umbilicus are commonly attacked, and it is there that a hard swelling is perceived, over which the skin is rather hot and sometimes red. The tumefaction gradually disappears by resolution, or else fluctuation becomes from day to day more distinct, showing that suppuration is taking place; and the pus being discharged, immediate relief follows, and the pain and febrile symptoms cease.

Now, the disease rarely runs a very acute course; it lasts at least a week or two, and often much longer. Where much of the muscle is involved, the complaint simulates peritonitis,—more, however, the partial than the general kind. Where the inflammation of the muscle is not extended, the resemblance to inflammatory affections of the organs lying underneath the point of tenderness is even greater than to inflammation of the peritoneum. Hepatitis, splenitis, and gastritis have been mistaken for the affection of the abdominal parietes. These errors can be avoided only by taking into account the absence of disturbed function of the suspected viscus; often, too, the peculiar swelling furnishes a clue to the real nature of the case. But as regards signs of disturbed function, we must bear in mind that these are produced occasionally by disorder of the adjoining viscera. Thus, we have jaundice in abscesses seated in the walls in the right hypochondrium.[1] Abscesses in the abdominal walls are sometimes symptomatic of a more distant lesion, as of caries of a rib.[2]

Can we distinguish, with anything like certainty, between abscesses in the abdominal walls and instances of partial peritonitis leading to *collections of pus in the peritoneal cavity?* I believe not; for in both there is a tumefaction; in both the general symptoms are much the same; and, as happens sometimes in peritoneal abscesses, the pus presses its way through the parietes of the abdomen. Yet whenever we find a swelling which has come on gradually, or has followed a blow or a kick on the abdomen, or a swelling which is very hard before fluctuation appears; whenever the softening of the tumor is immediately preceded by distinct chills, and the skin covering it is tense, and heated, or reddish; wherever there are no symptoms pointing to a partial peritonitis, as an attendant on visceral disease, or as a

[1] As mentioned by Habershon, Diseases of the Abdomen, 1878.

[2] Oppolzer, Wiener Medizinische Wochenschrift, 1862.

consequence of general peritonitis,—we may infer that the affection lies in the abdominal walls. But the skin is not always discolored or hot, and the beginning of the swelling is sometimes veiled in obscurity. In some instances I have seen, in which there was great doubt, the aspirator drew off a very offensive pus and broken-down material; and I looked upon this—as the sequence proved, correctly—as indicating abscess in the abdominal walls. Abscesses within the abdomen seated at the upper part, if not caused by abscess of the liver, are, as Bristowe points out,[1] largely due to perforation of one of the hollow viscera with circumscribed peritoneal suppuration.

But it is not every case of abscess in the walls which is attended with symptoms that render it likely to be mistaken for the results of inflammation. Sometimes the preceding tumefaction is so hard, or it is so long before the process of suppuration sets in, that the affection is more liable to be confounded with abdominal tumors. The most trustworthy points of difference are furnished by a study of the history of the case; by the slow growth of the tumor on the one hand, and its far more rapid growth on the other; by the rise in temperature and by the absence, or at all events the comparative absence, of signs denoting serious disturbance in one or several of the abdominal viscera. Then, in doubtful cases, the aspirator or the exploring needle will be of use. The fluid thus obtained shows, under the microscope, shreds of broken-down muscle and of areolar tissue, mixed, if suppuration have begun, with pus. Again, stress may be laid on the occurrence of chills preceding the softening of the mass. In some patients the inflammation is unaccompanied by any appreciable signs; it leads to gradual changes in the muscular fibres, which do not reveal themselves until the disorganized muscle gives way. The fibres undergo softening or a true fatty metamorphosis, and the slightest force suffices to produce a rupture. Not a few cases have been reported in which one of the recti muscles has been torn asunder during a fit of coughing. The seat of laceration is generally about midway between the umbilicus and the pubes, a little to one side of the median line; the rent fills with blood, occasioning a circumscribed swelling and rigidity of the abdomen. There is sometimes pain, with nausea, vomiting, and obstinate constipation. Nay, the symptoms have imitated so closely a strangulated ventral hernia as to have led to the performance of an operation.[2]

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[1] Lancet, Sept. 1883.

[2] Richardson's case, American Journal of the Medical Sciences, Jan. 1857. Further instances of this accident are given by Virchow, in the Würzburg. Verhandl., Band vii. The description of abscesses in the abdominal parietes I have

Rheumatism of the Abdominal Walls.—Occasionally rheumatism attacks the abdominal muscles, and gives rise to local signs similar to those of peritonitis. But the pain is not so constant, nor is it spontaneous, as in this disorder. It is also less affected by movements or by pressure. Deep pressure causes little or no more pain than slight pressure; and it is only when the muscles are placed on the stretch that the pain is severe, or sometimes, indeed, at all produced. The pain is often one-sided, or much more marked on one side, and we find no meteorism, and but slightly elevated temperature, and not the anxious countenance of peritonitis. Moreover, the attack is apt to happen in those of rheumatic tendencies, and there is concentrated, highly acid, scalding urine. Rheumatic peritonitis may supervene on rheumatism of the abdominal wall.

Abdominal Hysteria.—No disease simulates peritonitis more closely than hysteria. The abdomen may be extremely painful to the touch, swollen and distended with gas, fever may set in temporarily, and yet the whole disorder be purely hysterical. To illustrate:

An unmarried woman, twenty years of age, consulted me on account of extreme tenderness of the abdomen which had developed in a few days. The abdomen was swollen and tympanitic, and so sensitive that it would not bear the pressure of her clothes; the pulse was frequent; the skin dry; the tongue lightly coated; the bowels constipated; the countenance expressive of distress. Here was certainly a group of symptoms like those of acute peritonitis. But the absence of the wiry pulse, the comparatively slight fever,—slighter, certainly, than was to be expected from such general and great tenderness,—and the expression of countenance, arrested my attention. I found that the patient had had similar attacks previously; that they had come on sometimes shortly before, sometimes shortly after, her menstrual period; but that for several months her menses had ceased to flow. The abdominal tenderness was in reality, as she represented it to be, very great; yet strong pressure produced no more pain than the lightest touch. Nor was the pain increased by deep inspiration, or by coughing, or by extending the thighs. Taking all these circumstances into account, as well as her age and sex, and her nervous temperament, instead of treating her for acute peritonitis, cold-water injections, mild purgatives, and a mixture of assafœtida and valerian were employed. Under these remedies, all the symptoms of the apparent peritonitis speedily vanished.

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drawn from cases chiefly coming under my own notice, and from manuscript notes taken by Dr. J. K. Kane at the Philadelphia Hospital.

Yet all cases of abdominal hysteria do not pass off so quickly; sometimes they are much more persistent, or recur frequently. They are from the onset unattended with fever, or, as the thermometer shows, the fever is fitful and soon ceases. The absence of febrile excitement, too, especially if taken in connection with the several localized and more or less distinctly circumscribed spots of tenderness, enables us to distinguish between peritonitis and those instances of neuralgia of nerves supplying the abdominal parietes, to which women who are laboring under disorders of the uterus are so liable.

Colic.—As already stated, the pain of colic is paroxysmal, and not attended with fever, or with much, if any, tenderness; while the pain of an inflamed peritoneum is constant, and associated with the greatest tenderness and with fever. Cases of colic do indeed occur in which we find fever and some tenderness; but it is likely that in such cases the peritoneum is really in parts injected or slightly inflamed.

The same remarks are applicable to those severe paroxysmal pains which accompany the passage of gall-stones or of urinary concretions, or which occur at the menstrual periods. They are frequently spoken of as varieties of colic, and, as far as their discrimination from peritonitis goes, there is no difference,—it rests on the same grounds precisely; for when there is fever or tenderness on pressure, it is likely that inflammation has been set up in those parts in which, or in the neighborhood of which, the pain is felt. In the so-called uterine colic, an injection of the peritoneum has positively been demonstrated.

Chronic Peritonitis.—An acute attack of peritonitis may imperceptibly assume a chronic form. The fever gradually disappears, or at all events lessens; but the exudations into the peritoneal cavity, whether organized or not, remain, and so do some abdominal pain and tenderness. In this condition the patient may continue for many months, now and then a fresh inflammation starting up in the peritoneum and giving rise to acute symptoms, or an intercurrent severe diarrhœa leading to rapid loss of strength. Again, the disease may develop slowly, be latent from the onset, and may not attract attention until the abdomen swells. In all cases, no matter what their origin, if they last for any length of time, debility and emaciation become marked symptoms; hectic fever is observed; decided effusion in the peritoneum is generally noticed; the legs become œdematous; and the patient may present the symptoms of septic poisoning and die worn out. Where recovery takes place, the exudation into the peritoneal cavity is either discharged through adjacent viscera; or is gradually absorbed; or is transformed into tissue. When the disease terminates in this way, it is apt to leave its traces in a chronic

thickening and roughening of the peritoneum. A friction may be often felt. Chronic peritonitis of latent origin and leading to much thickening is sometimes found to attend cirrhosis of the liver or contracted kidney. Under no circumstances is chronic peritonitis likely to be an independent affection.

Chronic peritonitis may be confounded with affections of the liver attended by impediment in the portal circle; and what adds to the difficulty in diagnosis is, that the liver is apt to atrophy in chronic diffuse peritoneal inflammation. The greater and more diffuse tenderness, the evening exacerbations of temperature, the absence of marked dilatation of the abdominal veins, and the less extensive peritoneal effusion indicate the latter affection.

Chronic peritonitis is often found in connection with *tubercles* or with *cancer*. It then gives rise to very considerable abdominal enlargement, and it is with the diagnosis of abdominal enlargements that these forms of chronic peritonitis will be considered.

Diseases attended with Pain and Tenderness in the Right Iliac Fossa.

Appendicitis.—Inflammation of the appendix is pre-eminently the disease attended with pain and tenderness in the right iliac fossa. The appendix has an average length of four inches, and the diameter of a goose-quill. It lies in the right iliac fossa, but is variable in position. It points for the most part downward, or downward and inward. A. T. Bristow and Fowler[1] locate for it a central point by drawing a line from the anterior superior spinous process of the ilium to the median line, and placing the central point from two to two and a half inches within the anterior superior spinous process. From this central point the appendix will radiate in different directions. The usual location of the appendix is at the edge of the right rectus muscle below a line drawn from the centre of the umbilicus to the anterior superior spinous process. Appendicitis is essentially a disease of adolescence and of young adults. It presents itself clinically in these forms: acute catarrhal appendicitis; ulcerative and suppurative appendicitis; perforative appendicitis; chronic recurring appendicitis.

Acute catarrhal appendicitis may come on from exposure to cold and wet. Fowler cites two such cases. Much more generally it is an infectious process due to hardened fecal masses leading by the irritation they produce to exudations in which extraordinary development

[1] Appendicitis, Philadelphia, 1894.

of bacteria, as of the bacterium coli commune, takes place. It may also be caused by other infecting processes or micro-organisms. The far greater prevalence of appendicitis since the recent wide-spread epidemics of influenza suggests that this subtle poison, too, may act as an exciting cause. The disease may also result from vascular disturbances or torsion of the part. It is at the bedside always extremely difficult to say what cause has given rise to the attack.

Whatever the immediate cause, whether it be a quickly acting one, or, as is more common, have been silently working, the attack itself is generally sudden, and announces itself by acute abdominal pain, by tenderness in the right iliac fossa, by nausea and vomiting. The pain and the tenderness are very significant. The pain may be referred to the lower part of the abdomen, but it is very often referred to the umbilicus or to the epigastrium. It has, especially at first, the character of colic. It is soon noted to be associated with tenderness, which is chiefly manifest at or near McBurney's point. This corresponds to the outer edge of the right rectus muscle, and is most readily located by fixing a spot midway between the anterior superior spine of the right ilium and the umbilicus. The patient lies on his back, because to do otherwise increases the pain, and very often the right rectus muscle is somewhat tense, a fulness or a slight tumefaction can be perceived in the right iliac fossa, and there is some impairment of tympanitic resonance on percussion. Tenderness and swelling, as well as the shape of the appendix, may at times be recognized by deep pressure, and palpation of the appendix, as recommended by Edebohls,[1] may thus become of value. In some instances, and I have met with a number of them, the sensitiveness is not in the right but in the left iliac fossa. Again, the tenderness may be at the upper part of the appendix, below, but near, the gall-bladder. Tenderness is always a very important sign, and when it lessens both in degree and in extent it denotes decreasing inflammation. The nausea and vomiting disappear in the progress of the case, though vomiting may return should there be perforation. If the peritonitis become general, abdominal distention will be marked. Other symptoms met with in appendicitis are moderate fever, constipation, urine diminished and frequently containing albumin and indican.

Some cases do not begin so acutely, but are rather subacute. The complaint presents the following history and symptoms: The patient has been suffering for some time from constipation, or alternately from diarrhœa and constipation. He has a dull pain referred principally

[1] American Journal of the Medical Sciences, May, 1894.

to the iliac fossa, and radiating to the hips. When the iliac region is examined, it is tender to the touch, full and hard, and dull on percussion, while around the dulness there is a very tympanitic sound, if the intestine be much distended. Colicky pains occur from time to time, but are mainly confined to the lower portion of the abdomen.

No matter what the beginning, the case in its further progress exhibits varied features: it may end in resolution, and hardened fecal matter is often passed; or the tenderness in the iliac fossa may become greater, and vomiting, decided fever, and the marked signs of an extending peritonitis appear; or ulceration of the appendix may allow a discharge of extraneous matter into the peritoneal cavity, which produces violent general peritonitis, or an abscess forms that ruptures and perhaps leads to the same results; or, again, the bowel may become so paralyzed or so constricted that it can no longer propel its contents, and the patient dies with all the distressing signs of intestinal obstruction.

There are other terminations with which experience makes us familiar. The attack may end in a chronic appendicitis, indicated by persistent tenderness and some swelling, pain on walking, and often dyspeptic symptoms and depression of spirits; or the chronic inflammation may lead to a series of recurring acute attacks. Then as complications in appendicitis we may have thrombosis of the iliac vein, iliac phlebitis, post-cæcal abscess, fistula into the bladder or rectum, hepatic abscess.

There are two very important questions that always arise in appendicitis: Is there pus present? Has perforation occurred?

It is always difficult to determine the presence of *pus*, and there are no certain signs. Chills are generally absent; the temperature is of little value. The most trustworthy signs are very decided tenderness, a local swelling, marked rigidity of the right rectus muscle, and waves of pain in the affected region.

Perforation of the appendix is most often seen among healthy young men. It is found chiefly in the form of appendicitis that has been caused by seeds and concretions of various kinds, cherry-stones, and foreign bodies. In a certain proportion of cases the symptoms have been latent until the perforation happened. Its most constant and the first decided symptom is sudden, severe abdominal pain. This occurred in eighty-four per cent. of the cases which Fitz in his admirable essay has analyzed.[1] The pain is mostly at first in the right iliac fossa, and is followed by tenderness which gradually extends. It may be

[1] Transactions of the Association of American Physicians, 1886.

accompanied by a chill, but I have known pain absent where a chill was decided. Fever, with a temperature of between 100° and 102°, is next observed; but it is not constant, for I have met with a temperature nearly normal in a case in which a gangrenous perforation of the appendix was found.[1] A circumscribed resisting swelling in the right iliac fossa, which forms in from two to five days, with impaired resonance on percussion and with a sense of fluctuation from the abscess that develops, and disturbed micturition, establish the diagnosis. A rectal examination may aid us in detecting the tumor, but, as I know from experience, is not absolutely to be depended on as a means of recognizing the swelling or the pus that has formed. In the majority of cases general peritonitis begins from the second to the fourth day after the perforation. The cases that die from shock die before the second day; but, as a rule, the collapse comes on more slowly than in other forms of perforative peritonitis. Leucocytosis, Richardson tells us, is invariable in perforative appendicitis. Obliteration of the dulness over the liver and spleen is not as often found as in other forms of intestinal perforation.

A question that arises is whether we can distinguish inflammation of the appendix from an *inflammation of the cœcum*, both of which were formerly included under the name *typhlitis*. There is no certainty in the diagnosis. But these facts will often aid us greatly. Most of the cases of inflammation of the cæcum are due to impacted fæces, and the history of preceding long-continued constipation, a resisting elongated mass in the right groin, slight pain, and absence of fever, are very significant. Then, perforating inflammation of the cæcum is very rare, while perforation of the appendix is of frequent occurrence.

Much used to be said about inflammation of the loose areolar tissue around the cæcum, *perityphlitis*, and consequent abscess. But we now know that the abscess nearly always has its origin in disease of the appendix. The collection of pus may find its way into neighboring viscera, or be discharged externally, or become encysted, or the sac rupture and fatal peritonitis ensue. The tumefaction which the abscess occasions is generally very evident. When, however, the pus burrows under the iliac fascia, the swelling may be slight. But under such circumstances there appears a characteristic sign: the pain on moving the right foot is intense, because the iliac muscles become involved in the disorder. If the swelling be great, there may be œdema of the foot and numbness of the thigh, from the pressure

[1] Seen with Dr. Morton.

on the vein and nerves. Perityphlitis with marked swelling in the right iliac fossa may disappear without an abscess forming.

Chiefly on account of the pain and tenderness, acute appendicitis may be confounded with a number of diseases, prominent among which are colic; bilious colic; renal colic; acute cholecystitis; perforation of the gall-bladder; typhoid fever; ulceration of the lower part of the ileum; obstruction of the bowel; tumors of the kidney and abscesses in or around it; floating kidney; inflammation of the right ovary; extrauterine pregnancy; pelvic hæmatocele; pelvic peritonitis; tubercular peritonitis; abscess in the abdominal walls; psoas abscess; hip-joint disease; abscess of the liver; distention of the cæcum; cancer of the cæcum; pneumonia.

The sudden pain, the acute indigestion, the nausea and vomiting may cause appendicitis at its beginning to be mistaken for *colic*, especially for bilious colic, but the localization of the pain and particularly the tenderness in the right iliac fossa are very different. On the other hand, the jaundice that attends or follows *bilious colic* is not a symptom of appendicitis, and the pain of this does not radiate to the shoulder and the scapula. The same localization of the tenderness is of value in distinguishing *renal colic*, where the tenderness, if it exist at all, is most marked over Poupart's ligament. Moreover, rectal and vesical tenesmus and retraction of the testicle, common in renal colic, are very rare in appendicitis. Yet there are cases of appendicitis at its upper end that are very misleading, and, as in two cases I saw, one with Dr. Keen, the other with Dr. Dupont Smith, only to be recognized by the changing seat of the pain. In Dupont Smith's case tympany was a marked symptom.

Pain and tenderness in the right iliac fossa may be the cause of *typhoid fever* being confounded with appendicitis. But neither pain nor tenderness is great in typhoid fever; then the characteristic temperature record, the nervous symptoms, the diarrhœa, the eruption, furnish striking points of difference. Appendicitis may exist as a complication of typhoid fever, as we shall find while treating of typhoid fever.

Ulceration of the lower part of the *ileum* produces pain and tenderness in the iliac fossa. But, combined as the ulceration generally is with tubercular disease, the history of the case gives a clue to the nature of the malady. Moreover, diarrhœa occurs, and there is not present a tumefaction dull on percussion. Should, however, perforation of the bowel take place before the patient is seen, and general peritonitis come on, the diagnosis is not so readily made, because we are deprived of the decisive proof furnished by the swelling.

Another difficult diagnosis is at times that regarding *obstruction* of the bowel; the more difficult because appendicitis may become a cause of intestinal obstruction. In both there is pain; in both constipation; in both vomiting. But the pain in obstruction is not localized, or attended with such a significant seat of tenderness as McBurney's point; the constipation in appendicitis is not so absolute, and flatus passes; the vomiting in this disease occurs early, then generally stops; late vomiting is the rule in obstruction, and it becomes fecal. Though fever is not a marked symptom of appendicitis, there is generally some. *Acute intussusception* has a different history, and makes its appearance suddenly with such peculiar signs that, although it may likewise occasion a tumor in the right iliac region, it can be generally distinguished from appendicitis. Yet, where the latter leads to intestinal obstruction, the diagnosis is not always obvious; and tenesmus and discharge of bloody mucus from the rectum may also happen in appendicitis as well as in intussusception. Moreover, both are diseases to which the young are specially liable.

As regards *tumors of the kidney and abscesses in it or around it*, the situation of the swelling is not exactly in the ileo-cæcal region, or at all events it is not confined to this region. The mass of the tumor lies in the loin, or above the anterior termination of the crest of the ileum; and the urine contains ingredients, such as pus, or blood, or heavy deposits of urates or phosphates, which show that the secretion of the kidney is abnormal. Moreover, there is no intestinal disturbance or marked local tenderness, such as we find in appendicitis. In *floating kidney* the mobility of the displaced organ, the slight tenderness, the dyspeptic symptoms, and the throbbing of the abdominal aorta are very significant. The occurrence of attacks of severe abdominal pain, with vomiting and fever, may be misleading, but their frequent recurrence and the absence of localized swelling over the seat of the appendix are valuable signs.

An *inflammation of the right ovary* gives rise to pain and tenderness in the right iliac region, and to fever. But it is associated with disturbance of the uterine functions, with characteristic ovarian pain, and occasions no perceptible swelling. A tumor of the ovary or of the uterus may produce a visible tumefaction; but, springing as it does out of the pelvis, its exact seat, its bulk, its shape, the absence of marked intestinal symptoms, and a vaginal examination, will permit its cause to be discovered. In *acute salpingitis* there is the history of infection, absence of vomiting, and but slight degree of abdominal tenderness and rigidity.

Extrauterine pregnancy may be mistaken for acute appendicitis in

consequence of the sudden rupture of a sac. But the previous history, the great prostration, the excessive thirst, and a pelvic examination will explain the true meaning of the symptoms. In *pelvic hæmotocele* the pain and the suddenness of the attack make us think of acute appendicitis. But the tumor that forms is generally larger, doughy; there are no localized spots of tenderness, no marked intestinal symptoms; and the history of irregular menstruation and a vaginal examination will remove all doubt. Ovarian cysts with twisted pedicle, ovarian abscess, pyosalpinx, fibroid tumors, a varicose condition of the veins of the broad ligament, and painful menstruation may also be mistaken for appendicitis; but as Deaver,[1] in an admirable paper based on extraordinarily large experience, shows, none has the exact combination of signs found in appendicitis. This may exist as a complication of pregnancy.

Generally in *diseases of the gall-bladder* the seat of pain and tenderness is over it, and not in the right iliac fossa, as in appendicitis. But there are exceptions in both affections, rendering the diagnosis very difficult, it may be impossible. The swelling of a distended gall-bladder may be felt very low down, and, on the other hand, appendicitis of the upper part may have its local signs in the neighborhood of the gall-bladder. Rigidity of the rectus muscle and pain are common to gall-bladder disease and to appendicitis. The pain of acute cholecystitis is, however, more violent; and so it is, as a rule, in perforation of the gall-bladder than in perforative appendicitis. Still, how deceptive symptoms may be is proved by the published cases of eminent surgeons like Fowler[2] and like Richardson.[3]

An *abscess in the abdominal walls* furnishes very many of the signs of abscess around the appendix. The most trustworthy point of distinction is that the former moves with the abdominal walls and is unassociated with intestinal irritation, while the latter is commonly so combined. Then the peculiar spots of tenderness, the outline of the swelling, its want of prominence, are unlike what is found in abscess of the abdominal walls.

In *psoas abscess* we have the association with caries of the vertebræ: rigidity or an excurvation of the spine, dorsal pain and tenderness, testify to this connection. It occurs in scrofulous persons, and, although gradual in its formation, is often sudden in its manifestation; for not unusually a fluctuating, painless tumor appears below Pou-

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[1] Appendicitis in Relation to the Diseases of Uterine Adnexa and Pregnancy, Medical News, Oct. 1897.

[2] Op. cit.

[3] Amer. Journ. Med. Sci., July, 1898.

part's ligament as the first positive sign of this formidable affection. This is very different from the history of an appendicitis which has led to post-cæcal abscess. Moreover, preceding the pointing of the psoas abscess at the spot mentioned, there are often indications of irritation in those muscles in the sheath of which the pus travels; there is difficulty in extending the leg, with inability to stand upright.

Pelvic peritonitis is not likely to be mistaken for appendicitis, except in those rare cases in which the appendix is lodged in the pelvis. Treves[1] mentions such a case. He also cites one of *tuberculous peritonitis* the cause of error. While the local signs may be misleading, the previous history, the amount of fever, and the grave constitutional symptoms are likely to aid us to a correct conclusion. In *hip-joint disease* the inclination of the pelvis, and the inability to move the joint normally, furnish trustworthy points of distinction.

It is sometimes difficult to distinguish between appendicitis, especially in its chronic forms, and *abscess of the liver;* the more difficult because, as I know by experience, they may coexist, the hepatic abscess being consequent to the appendicitis. Another fact that makes the diagnosis difficult is that the pain and tenderness in appendicitis do not always exist in the right iliac fossa, but may be found at various parts of the abdomen; the abscess following appendicitis may extend high up towards the liver. In these difficult instances the history of the case, as well as the study of the sequence in which the phenomena appeared, becomes of the greatest value.

A *distention of the cœcum* may be mistaken for chronic appendicitis. It gives rise to fulness in the right iliac fossa, and to pain, often of colicky character, but, unless associated with inflammation, not to tenderness or to fever. Purgatives, too, clear out the fæces which accumulate from want of power of the bowel to propel them, and the dulness on percussion vanishes after the free evacuations, and, except when the cæcum is loaded with fæces, it is highly tympanitic.

In that rare disease, *cancer of the cœcum*, there is a fixed, firm swelling; but it is of very gradual growth, and the disorder generally produces a stricture of the bowel and is associated with malignant disease in other parts of the body.

Other affections than those of the bowels may give rise to signs supposed to indicate appendicitis. It does not at first sight seem likely that this would be the case with *pneumonia*. Yet the mistake has been committed. Pain is sometimes referred to the right groin in pneumonia, and there is soreness there, connected probably with the

[1] Allbutt's System of Medicine, vol. iii., article "Perityphlitis."

efforts at coughing and the disordered breathing. Nay, I have known poultices to be applied to the right iliac fossa to relieve the inflammation which really was in the chest. An examination of this part of the body will, of course, at once explain the true character of the symptoms.

Hysteria may take on the form of appendicitis, but there is no accurately localized tenderness and swelling, nor fever. The wide discussion of the subject of appendicitis and the popular interest taken in it have led to a new form of hypochondriasis.

In chronic appendicitis there is at times a strong tendency shown to recurring acute or subacute attacks. In one instance that came under my observation there were forty-seven before the case was operated on. Generally in these cases of *recurring appendicitis* a chronic thickening of the appendix is present with or without adhesions, and the tube is narrowed or obliterated; there is obliterative appendicitis. An induration may nearly always be felt in the region of the appendix, and there is tenderness on deep pressure, and mostly some impairment of general health and symptoms of intestinal dyspepsia. Indiscretions in diet or active exercise is very apt to bring on an acute attack, and perforation may be the outcome of many.

Disorders attended with Constipation, and of which it is a Prominent Symptom.

An inactive state of the bowels is often but a concomitant of some disorder which presents much more striking phenomena. But there are cases in which the constipation is the most important symptom, and in which it furnishes decisive proof of a morbid condition of the intestine. Now, these cases are either those in which the constipation arises suddenly, or at any rate becomes suddenly aggravated, and is often insuperable; or those in which it is an habitual state, and is not associated with any signs of urgent distress.

Intestinal Obstruction.—Intestinal obstruction, when coming on suddenly, manifests itself generally in the following manner: A person, previously in good health, or perhaps of costive habit, notices that his bowels have not been moved for several days, and that he has an uneasy feeling in the abdomen in consequence. He takes the purgative he is wont to employ, but without the usual effect. Something more active is tried, and still the bowels remain obstinately bound. Severe colicky pains have in the mean time made their appearance. He becomes alarmed, and sends for his physician, who sees that there is indeed cause for alarm. The abdomen is found to be distended, but not painful, or only slightly painful, on pressure. But through its

parietes may be noticed the violent, rolling motion of the irritated intestine. Vomiting sets in,—first, of the substances contained in the stomach or of a bilious fluid, and, as the case progresses, of stercoraceous matter. In this way, unless nature or art comes to the rescue, the disease continues; and signs of inflammation of the bowels, and with them fever, appear as preludes to the fatal termination. Sometimes, however, the patient becomes gradually exhausted; there are no tenderness and fever, but a cool skin, a quick, small pulse, a countenance ghastly and panic-stricken. Violent paroxysms of pain, alternating with intervals of ease, may occur to the last moment. But, in spite of the utter prostration, the mind generally retains its clearness. Should recovery take place, large quantities of fecal matter are discharged, and the symptoms of the impediment speedily disappear.

These phenomena are too striking to permit of errors in diagnosis. Yet errors are of frequent occurrence, because the history of the attack and the sequence of the symptoms are not taken into account. Many a person laboring under *peritonitis* has been violently purged to remove the stubborn constipation believed to be due to a mechanical hinderance in the bowels; and, on the other hand, many a case of intestinal obstruction has been treated solely with reference to the inflammation that may attend it, and without regard to the source of the inflammation. Yet it is not ordinarily difficult to distinguish which is cause and which effect. A case that begins with severe colicky pains and obstinate constipation, in which, at first, in spite of the pain, there is little or no tenderness; in which the temperature is normal or subnormal; in which vomiting and tympany soon occur; in which fulness on palpation and dulness on percussion may be detected at or above the point of stoppage; and in which fecal matter is ejected by the mouth after a stoppage of the bowels of a few days' duration,—is not primarily, whatever may be the ultimate complications, enteritis or peritonitis. A case presenting almost from the onset fever and great and extended tenderness, in which vomiting of fecal matter, if it happen at all, does not happen until late; in which diarrhœa is sometimes found to supersede the enduring constipation,—is inflammation of the peritoneum, but not a mechanical obstruction. Only in rare instances, and especially when the bowel is invaginated, is the malady so quickly succeeded by inflammation as seemingly to make its appearance with the signs of peritonitis. Perforative peritonitis, with its signs of collapse, shows a much stronger likeness to acute obstruction of the bowel than ordinary peritonitis does.

The symptoms dwelt upon as pointing to an intestinal obstruction

bear a close resemblance to those of *external strangulated hernia*. In truth, they not only resemble but are identical with those of this affection. Hence in every case of obstinate constipation each point which may be the seat of a hernia must be explored by the eye and the hand. No motives of false delicacy, no reluctance, should prevent the physician from insisting on a search, the neglect of which may cost a life.

It would be foreign to the object of this work to discuss the external signs by which a strangulation of the intestine at a hernial opening manifests itself. It need only be mentioned that it is at the groin, at the umbilicus, at the side of the anus, or through the ischiatic notch that the gut descends and forms a tumor, and that these are, therefore, the regions to be scrutinized. Moreover, there are internal hernias that become strangulated, such as a diaphragmatic hernia, a hernia into the foramen of Winslow. But these are matters more strictly surgical. Yet there is one part of the subject, of importance alike to the physician and to the surgeon, which cannot be passed by without a few words, since it may be a cause of much perplexity,—namely, the possibility of intestinal obstruction taking place in a person laboring under an irreducible hernia and simulating strangulation without any strangulation having occurred. Of this the following case furnishes an example.

A number of years since I was requested by a physician to see with him a woman, the mother of thirteen children, who had been for days laboring under obstinate constipation. Large doses of mercurials, croton oil, and turpentine enemata had failed to procure a passage, and the patient was becoming much frightened. Nor was her situation free from danger. She had considerable pain in the abdomen; she had been vomiting stercoraceous matter profusely; the rolling of the intestines could be plainly perceived. On her right side was a small irreducible femoral hernia, which had existed for years. It was not painful on pressure, nor was the skin discolored; neither did the mass itself communicate an impulse during the act of coughing. Here were signs of a serious impediment to the onward passage of the intestinal contents, as the fecal vomiting and the rolling of the intestines showed plainly. But was it due to strangulation at the hernial opening? Was it an internal intestinal obstruction?

An accurate examination of the abdomen did not throw much light on these questions. The belly was moderately tympanitic, and not painful to the touch, except when the pressure was considerable. The rolling of the intestines was perhaps more obvious on the left side; but nowhere could a tumor be felt. Taking all the circum-

stances of the case into account,—the fact that the patient was of costive habit; that she was subject to attacks of colic and of obstinate constipation; that there was nothing to prove that the hernia had recently increased, or was in any way inflamed,—the conclusion arrived at was that the case was not one of hernial strangulation, but of internal intestinal obstruction. Copious warm-water injections were thrown into the colon through a flexible tube; her abdomen was rubbed with mercurial ointment. But all in vain: she continued vomiting fecal matter.

Her situation now appeared desperate. She had not had a passage for six days; she was steadily sinking. Knowing that sometimes the gut may be strangulated at a hernial opening without much pain or tenderness, the counsel of an eminent surgeon was sought, to aid in determining whether this was not the cause of the impediment. He thought it probable that it was. The patient was etherized, and the hernial section performed; but no constriction was found. The wound was closed, and large doses of opium were administered, so as to mitigate, so far as practicable, the torture of the only termination to the case which seemed possible. On the day after the operation, the intestines had ceased to roll; there was no vomiting. But stercoraceous vomiting reappeared two days afterwards, and the rolling of the intestines was occasionally, although faintly, perceptible.

The patient's exhaustion was now extreme; her pulse was very quick and small; her skin cold, of a dirty look; the odor of the breath and of the whole body offensive; and the eyes sunken and surrounded by a broad leaden ring. There was slight pain on pressure between the umbilicus and the sigmoid flexure. The vomiting had ceased, or occurred only occasionally. Although there was little hope, we had, as soon as admissible after the operation, recommenced rubbing mercurial ointment over the abdomen, and giving injections in the manner before described. This was continued until, to our great gratification, one morning, after a tube had been passed a distance of several feet into the colon, the patient had a copious discharge of tarry fecal matter from her bowels,—seventeen days after the symptoms of complete intestinal obstruction had declared themselves by the occurrence of stercoraceous vomiting.

This case is instructive in more than one respect. It teaches that recovery may take place most unexpectedly after many days; and, in a diagnostic point of view, it illustrates a difficulty which any physician may have to encounter in attending a patient the subject of a long-standing hernia.

Supposing that the symptoms are altogether owing to an obstacle

at some portion of the intestine within the abdomen; can we determine the exact position of the impediment and its nature? We know how varied are the conditions which lead to sudden and invincible constipation. We know that strangulation from bands and adhesions, or gaps in the omentum, or the pedicle of an ovarian tumor; that intussusception; that twists and knots; that strictures and tumors; that abnormal contents, such as foreign bodies, impacted fæces, gall-stones, worms, concretions of drugs, as of bismuth, may all occasion intestinal obstruction. We also know that in certain cases the obstruction is from spasmodic contraction of the intestine,[1] or paralysis of the bowel. Can we distinguish these different lesions at the bedside? In certain cases we can,—we can determine exactly both the position and the character of the lesion; in others there is no clue to an accurate discernment of either. It is possible that in time the X-rays may give us the desired information.

Obstruction of the bowel may present itself as an *acute* or as a *chronic* malady. The same symptoms occur in both. It is the mode of origin that is different. Nay, the same lesion may occasion in some instances an acute, in others a chronic, affection. Intussusception, internal strangulation, volvulus, impaction of a large gall-stone, are generally acute; strictures, tumors, contractions, and, for the most part, fecal accumulations, lead to chronic obstruction. Then there are cases that pursue a chronic course, but which terminate in acute obstruction. In *acute* intestinal obstruction, the first marked symptom is violent abdominal pain in the region of the umbilicus; there are early and persistent vomiting which becomes stercoraceous, great thirst, and often speedy collapse. Unless peritonitis supervene, we find no fever; towards the end the signs of septic poisoning may show themselves. In chronic intestinal obstruction the pain is at first like ordinary colic, and gradually becomes more persistent; nausea is almost constant, but vomiting is not, except towards the end, a pronounced feature, and the constipation only gradually becomes absolute. The abdomen is distended and the seat of gurgling sounds, tenesmus is common, and a tumor, often the result of fecal accumulation, can be felt. The breath acquires a fecal odor; the appetite utterly fails. Unless the obstruction can be relieved, the patient dies worn out, and from ptomaine-poisoning.

We shall first examine the more common kinds of the *acute* form. Among these, *intussusception* or *invagination* is frequent and at the same time the least difficult of recognition. Part of the bowel

[1] Archives Générales, Aug. 1868; Flint, Practice of Medicine.

becomes inverted, slipping into the cavity of the adjoining upper or lower portion. Inflammation is soon set up, produces infiltration of the tissues, and often leads to adhesions between the opposed serous surfaces. The inflammation may spread rapidly over the serous membrane, and the patient may die from general peritonitis. But sometimes in the inflammation that is lighted up at the seat of the ileus lies safety. It may give rise ultimately to a sloughing off of the invaginated part and its discharge into the bowel, while the mass of adhesive lymph surrounding the seat of ulceration maintains the continuity of the intestinal canal; thus the inflammation may pave the way to a favorable issue by restoring the caliber of the tube,—sufficiently, at any rate, to permit of the transit of its contents.

When the intussusception takes place rapidly, a sudden local pain is produced, recurring in paroxysms, and likely to be referred to the seat of the disturbance. The pain is quickly followed by vomiting, by constipation, by tympany, and by tenderness. But the constipation is not so absolute as in other cases of intestinal impediment; is, indeed, often preceded by diarrhœa. Not unusually, owing to the invaginated bowel remaining open, the liquid contents of the intestine pass through the intussuscepted part and produce a deceptive diarrhœa; yet oftener occur tenesmus and discharges of bloody mucus. Both of the latter signs are eminently diagnostic of the lesion. Still more so is feeling the end of the invaginated bowel by an exploration of the rectum, or finding the loosened segment in the stools. But it is only in cases in which the lower portion of the canal is affected, or which have been sufficiently protracted to allow of the curative efforts of nature being accomplished, that signs so pathognomonic are met with. Vomiting is not a marked feature of acute intussusception; it sometimes passes away and returns. The tenderness at first is localized, but spreads as peritonitis spreads; there is rarely tympany.

The casting off of the sloughed portion of the intestine is attended with hemorrhage. Whether this be the only cause of the hemorrhage or not, it is undoubted that purging, or sometimes vomiting, of blood, is among the differential signs of intussusception. A sign more valuable, because so much more usual, and present in about half the cases, is a tumor, frequently of cylindrical shape. Its seat varies with the seat of the lesion; and as the most common invaginations are those of the ileum and cæcum into the colon, or those at the inferior portion of the ileum, it is at the lower part of the belly, and in the right iliac fossa, that the swelling is detected. In the attacks of pain, the tumor becomes harder and larger. When low down in the

rectum, or protruding from it, it has been mistaken for hemorrhoids or prolapse of the bowel.

The malady is generally due to irregular peristalsis; it is sometimes caused by tumors of the intestines, particularly by lipoma.[1] The majority of cases of invagination happen in children under ten years of age, and a number are met with in infants. The course the affection pursues is rapid; the patient dies generally in less than a week after the occurrence of the accident, utterly prostrated. The cases which get well recover either gradually after the invaginated bowel has been discharged, or, in rare instances, quickly by the inverted bowel righting itself.

Acute obstruction from *internal strangulation*, as by bands or through apertures, is almost invariably seated in the small intestine. Its most characteristic feature is furnished by the history of a previous peritonitis, an operation on the abdomen, or an appendicitis. There is rarely fever; the obstruction has a sudden onset and soon becomes complete; nausea and vomiting set in early; fecal vomiting usually begins from the third to the fifth day. It is the decided exception to find a tumor; tympany may or may not be marked, but no flatus escapes by the bowel. Of further significance in the diagnosis of internal strangulation are the occurrence of collapse almost from the beginning; the frequency with which the disease is found in young adults; the rapid course it runs; the severity of the pain, which is generally referred to the umbilicus; the intense thirst; the absence of external or of discoverable obturator hernia; the absence of visible peristole,—such as happens in stricture,—of tumor, of hemorrhage, of tenesmus, and of dysenteric symptoms, as seen in intussusception. Obstruction by a band connected with a diverticulum scarcely ever occurs except in males under twenty years of age.[2]

Acute obstruction from *volvulus* or twist begins with severe abdominal pain, which soon becomes associated with nausea and vomiting and extreme distention; it rarely presents a tumor or visible intestinal coils, or elevation of temperature.[3] It nearly always affects the sigmoid flexure, and is preceded by a history of constipation; the pain at first is intermittent. There is local tenderness over the distended colon, also tenesmus; vomiting may be absent. The meteorism is very great, and peritonitis soon becomes a complication. The

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[1] Clos, De l'Invagination intestinale, etc., Paris, 1883.

[2] Fagge, Practice of Medicine, vol. ii.

[3] Fitz, 'Acute Intestinal Obstruction, Transactions of Congress of American Physicians and Surgeons, vol. i., 1889.

constipation is absolute. Tenesmus and dyspnœa are not infrequent. It is commonly met with in men after forty years of age.

Obstruction by a large gall-stone is apt to occasion severe attacks of colic as the gall-stone passes along the course of the small intestine, and is temporarily arrested. There is also vomiting, but no decided abdominal tenderness, and no tumor. Similar but less severe symptoms may happen, with periods of entire relief, until the gall-stone becomes impacted, and the constipation absolute. We shall be greatly assisted in the diagnosis by the history of previous biliary colic, particularly if the symptoms are met with in a fat, elderly woman.[1] The signs of intestinal stone, or *enterolith*, are those of a gradual and chronic, and not of an acute, obstruction. Obstruction from the swelling of a foreign body that has become impacted in the intestine can only be discriminated by the history.

There are other and rarer forms of lesions than these discussed as leading to acute obstruction, especially connected with the different results of adhesions and matting together of the intestinal coils, but there is nothing in the symptoms to guide us in deciding on the exact nature of the obstacle.

Chronic obstruction of the bowel is generally produced by fecal accumulations, by chronic intussusception, or by strictures. Chronic obstruction from *fecal accumulations* occurs chiefly in women, especially neurotic women. There is the history of a long-standing constipated habit, with attacks, perhaps deceptive, of catarrhal diarrhœa, produced by the irritation from the hardened fæces, with offensive breath, at times with slight fever. Pain and vomiting occur as late symptoms, and a tumor or tumors are noticed in any part of the large intestine. The tumor is usually painless, and has a doughy feel; the abdomen is very distended; but, except the occlusion be low down in the descending colon, there is no tenesmus. The constipation gradually increases, and, unless relieved, becomes insuperable. Fortunately, it generally can be relieved.

Chronic intussusception may extend over months. The symptoms are much the same as in the acute form, save that tenesmus is less common. Tumor, as in acute intussusception, is present in about half the cases. Paroxysmal pain and diarrhœa are generally prominent, vomiting is not. Blood is frequently passed with the stools. The patient is apt to die from exhaustion.

Strictures of the bowels are generally cancerous. They mostly occur after the age of forty, and are of slow development. The ob-

[1] Fagge, Practice of Medicine, vol. ii. p. 210.

struction is shown by the alteration in the shape and size of the fecal discharges, which become flattened. But this is far from an invariable rule. In the majority of cases the stricture is at the sigmoid flexure, and often at its lower part. There are paroxysms of pain, distention of the abdomen, and attacks of constipation that become more and more protracted until obstruction occurs, unless death take place previously from the cachexia. Vomiting happens only as a late symptom. Tenesmus, bloody discharge from the bowel, and hemorrhoids are often met with. Treves[1] states that tenesmus is more marked early than late in the disease. In malignant cases we can generally feel a tumor through the abdominal walls. If in addition to the symptoms enumerated, a bougie passed into the rectum meet in its course with a decided obstacle, an error in diagnosis is hardly possible. When, however, the stricture is not accessible to instrumental examination, although we can commonly recognize its presence, we cannot fix its site. The distention above the narrowed part is often so extreme as to lead to displacement of the colon and to an almost uniform swelling of the whole abdomen. For instance, in a case reported by Albert H. Smith, the enormously dilated colon had broken loose from its attachments and concealed the rest of the viscera. It was in several places eighteen, in none less than fifteen, inches in circumference.[2]

Other causes of stricture besides cancer, though less common ones, are cicatrization of extensive syphilitic, tuberculous, or dysenteric ulcers. Save in the tuberculous form, there is not marked cachexia, and a tumor can rarely be felt. Obstruction produced by the pressure of tumors external to the bowels cannot be distinguished from that due to intestinal stricture, except it be by the antecedent circumstances. In all cases of *constriction of the small intestine*, however caused, there are signs of indigestion and colicky pains. Tenesmus does not happen, but vomiting is more common than in stricture of the large bowel. A contraction in the small intestine is seen chiefly as the result of chronic peritonitis binding down the bowel, and may lead, like a stricture, to chronic obstruction.[3] *Fecal accumulations* also produce chronic obstruction.

With reference to the *frequency* of the different forms of intestinal obstruction, the elaborate studies of Fitz[4] give us valuable informa-

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[1] Article "Intestinal Obstruction," Allbutt's System of Medicine.

[2] Proceedings of the Pathological Society of Philadelphia, Dec. 1858, vol. i.

[3] Fagge, Guy's Hospital Reports, 3d Series, vol. xiv.

[4] Transact. of Congress of Amer. Phys. and Surg., vol. i., 1889.

tion. Strangulation is the most frequent cause of acute obstruction, occurring in fully one-third of the cases; a number are noted as following operations upon the pelvic organs in women, though the disease is very much more common in men than in women. Intussusception comes next in frequency, and is especially seen among children and young adults. Volvulus or twist is mostly encountered in men, and in half the cases is in the sigmoid flexure. Strictures and tumors, that are such usual causes of chronic obstruction, very rarely lead to acute obstruction. Treves,[1] from an examination of the records of the London Hospital, regards the cases due to fecal accumulation as the most numerous, and those caused by intussusception as more common than those from strangulation.

In any kind of obstruction the *location of the lesion* is difficult to determine. There are, however, a few circumstances which may aid us in arriving at such a determination: one is the fact pointed out by Barlow,[2] that the higher up the obstruction is in the canal, the nearer therefore to the stomach, the smaller is the quantity of urine passed; another is the early and more persistent occurrence of the vomiting and its want of stercoraceous character,—both of which render it likely that the impediment is in the small intestine and remote from the cæcum. Another is the early presence and the greater severity of hiccough when the mischief is in the small intestine, and the greater constitutional disturbance. Another is the absence of tenesmus except in acute intussusception. Yet another, that by far the largest number of cases of acute obstruction have the lesion in the small intestine, while in the chronic ones it is generally in the large bowel. Sometimes the patient is himself aware of the exact seat of the cause of his suffering; he notices that the injecting tube or the enemata seem to reach a certain point and go no farther; so, also, with the rumbling of the wind. Again, these borborygmi are especially apt to occur in obstructions of the large intestines, and, if joined to tenesmus, are signs of some importance. Indican is found in the urine in greatly increased quantities in stoppages of the small intestine. We may also be able to come to some conclusion about the seat of the lesion by finding out how many quarts of warm water we can inject into the large intestine.

The location of the pain, too, may furnish a clue to the position of the impediment. If this be in the small intestine, the pain is apt to

[1] Loc. cit.

[2] Guy's Hosp. Rep., 2d Series, vol. ii. Brinton accepts this statement only in so far as the amount of vomiting, which is apt to be greatest when the obstruction is high up, influences the amount of urine passed.

be chiefly, if not entirely, in the neighborhood of the umbilicus. Another circumstance on which some stress may be laid is the distention of the intestine above the point of intussusception. Indeed, this distention may occasion a visible fulness, sounding extremely tympanitic on percussion; at times, too, a slight dulness is found, attended with some resistance at or immediately above the seat of the obstruction. But neither the swelling nor the tympanitic dilatation of the bowel—as William Brinton[1] has proved—is a certain sign; indeed, with the exception of a tumor dull on percussion and resistant to the touch, there is nothing absolutely indicative of the lesion being at a particular spot. It is hardly necessary to say that a swelling of this kind cannot always be found.

Pain and swelling in the right iliac fossa may be caused by an *appendicitis*, and the constipation which may attend is most obstinate and in some instances incurable, causing the disease to enter into the category of intestinal obstructions. We have already, when treating of appendicitis, discussed the diagnosis between this and intestinal obstruction. It is in appendicitis important to note that should the constipation have become unyielding, the tumor and the other local signs do not follow the insuperable constipation, but precede it. Stress may be laid upon the occurrence of the signs of collapse in *perforative appendicitis*, though these may be slow in their development. In *acute hemorrhagic pancreatitis* there may be also the signs of intestinal obstruction, not to be distinguished except perhaps by the history, the extremely rapid course of the disease, and the marked peritonitis.

Symptoms like those of intestinal obstruction may also result from *occlusion of the mesenteric arteries* by thrombosis or embolism, in consequence of atheroma or inflammation of the vessels, arteriosclerosis, or valvular disease of the heart. There may be besides severe abdominal pain, vomiting that may become stercoraceous or bloody, tympanites, and signs of collapse. Instead of constipation or in its sequence there may be diarrhœa, with bloody stools. Sometimes a tumor may be palpable. The affected portion of bowel may undergo ulceration or gangrene.

Habitual Constipation.—This is a chronic state, unattended with urgent symptoms of any kind. Yet it is an annoying and very prevalent complaint. The symptoms encountered, independently of the rare and difficult fecal evacuations, are headache, giddiness, sluggishness of mind, a want of the natural appetite, anæmia, cutaneous

[1] Croonian Lectures, and work on Intestinal Obstruction.

eruptions, and, joined as the disorder not infrequently is to derangement of the stomach and of the biliary secretion, digestive disturbances and a sallow complexion: an altered state of the blood from the absorption of ptomaines may exist. In women there are also often added to the list of evils to which costiveness gives rise, neuralgic pains, palpitation of the heart, cold feet and hands. Infrequent evacuation of the bowels does not always produce such unpleasant consequences. It may, indeed, in individual cases be compatible with perfect health; for what is costiveness in one person may be a natural state in another.[1]

Habitual constipation is produced by various causes. It may be brought about by the peculiar nature of the diet. It may depend upon a deficiency or a faulty composition of the intestinal secretions, or upon disorders of those neighboring glands which pour their secretions into the intestines. It may result from impaired power of the bowel to propel its contents, the consequence either of some mechanical interference with its action, or of nervous influences, or of exposure to the poisonous effects of certain substances, as of lead. To particularize the numerous conditions which furnish illustrations of each of these different causes would serve no useful purpose. A few only need be specially noticed.

We have often to treat constipation in those who are dyspeptic and suffer from piles. In them there is, in all probability, some congestion of the portal system, and not infrequently a constant derangement of the flow of blood through the liver. The normal secretion of intestinal juices is interfered with, healthy bile is not supplied, and costiveness results. A similar congestion of the intestinal mucous membrane has its share in producing the constipation which is encountered in disease of the heart. Sometimes, however, enough healthy fluid is poured out within the intestine; but the inclination to go to stool is resisted, and the liquid that has been mixed with the matter to be voided is reabsorbed.

The influence of the nervous system on the alimentary tube is shown by the confined state of the bowels which attends excessive intellectual exertion and violent emotions. And when these states are protracted, they lead to a permanent and annoying debility of the intestine. The colon especially becomes torpid in its action, and all the evil results of constipation show themselves in the most marked degree. Not that an atony of the bowel is always due to psychical

[1] In the American Journal of the Medical Sciences, Oct. 1874, a case is reported in which the constipation lasted eight months and sixteen days.

agencies. Any disorder which induces loss of power in the muscular fibres may give rise to it. We find it in anæmic persons and in those who lead, so far as bodily exertion is concerned, a sluggish life. In some cases—fortunately rare—the weak intestine distends greatly, and becoming unable to propel the accumulated fæces, insuperable constipation occurs. The same complete paralysis of the tube may be brought about by chronic lesions of the brain or spinal cord.

Among the different organic changes in the intestine which, by interfering *mechanically* with the peristaltic wave, set up constipation, we find distention of the tube, with atrophy of the muscular fibres; various infiltrations into the walls, producing a narrowing of the caliber, as in carcinoma; and adhesions between the serous coats of the intestines, or between these viscera and the parietes. Of the first, it need only be said that the symptoms are due to the same paralyzed condition of the intestine, whether complete or incomplete, which has been already considered, and which is recognized, so far as it can be recognized, by the history of the case. The second group embraces those infiltrations which result from inflammations, and new growths of different kinds which lead to strictures, and then the peculiarities in the form and size of the fæces, the gradual wasting and exhaustion, and the extreme costiveness, deepening gradually into invincible constipation, furnish a key to the grievous nature of the affection.

When the constipation arises as the result of peritoneal adhesions, there are sometimes signs in the case—such as tenderness at a particular spot from still existing inflammation, or partial distention or retraction of the abdomen—which point out its nature. In the absence of these, the history is our only guide, except in those instances in which, as Bright[1] first informed us, a peculiar sensation is communicated to the touch, varying between the crepitation produced by emphysema and the feel derived from bending new leather in the hand.

From long-standing constipation *stercoral ulcers* may arise. The sacculi of the colon are filled with little, hard, fecal balls, which irritate the mucous membrane and produce ulceration. Mucus, or mucopus, with stains of blood, is occasionally discharged with the small scybala, and at times there is diarrhœa.

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[1] Cases Illustrative of the Diagnosis of Adhesions and other Morbid Changes of the Peritoneum, Med.-Chir. Transact., vol. xix.

Disorders in which Morbid Discharges from the Bowels occur.

Matters very unlike the healthy alvine evacuations are often voided from the intestinal canal: loose watery stools, large quantities of mucus, pus, or blood, may be discharged. The disorders which occasion these discharges may be here described.

Diarrhœa.—Like constipation, diarrhœa will be merely treated of as we meet with it constituting the entire ailment, or at all events its most prominent symptom. There are several varieties of diarrhœa. Difference in time gives rise to marked varieties,—to an acute and to a chronic form; and of both it has been already pointed out how often the lesion is an intestinal catarrh.

Acute Diarrhœa.—Acute diarrhœa proceeds from more than one cause: it may be excited by the irritating character of the food taken, or by impure water; it may be brought about by the morbid nature of the secretions poured into the intestines; it may be owing to atmospheric influences,—to heat, to moisture, to contaminated air; it may be caused by chilling of the surface of the body, or by irritant poisons, retained fæces, or worms. It may be occasioned by pyæmia and septicæmia, by reflex irritation, as in dentition, or by mental emotions, and especially by fear. Sometimes it occurs in an epidemic form due to some unknown miasm. Its symptoms are thirst; abdominal uneasiness; griping pain in the bowel; pallor; slight debility; and frequent fluid alvine evacuations, which may finally become almost colorless.

In the diarrhœa caused by a debauch or by indigestible food, nausea and a furred tongue are added to the list of symptoms mentioned. This kind of diarrhœa is generally of short duration. It is an effort of nature to get rid of obnoxious matter; and when this is effected, the looseness of the bowels ceases.

The variety of diarrhœa under consideration sometimes goes hand in hand with a disturbance of the biliary functions, and the stools discharged are fetid, and present the appearance generally described as bilious. This "bilious diarrhœa," too, is not uncommon in persons whose livers are habitually sluggish. It is also frequently encountered during the hot months of summer and early in the autumn, and has a tendency to run on.

There are cases of diarrhœa attended with pain, considerable soreness to the touch, and, what is not ordinarily met with in diarrhœa, some febrile disturbance. These kinds of acute diarrhœa, or rather of acute intestinal catarrh, or of muco-enteritis with diarrhœa

as a symptom, are often the consequence of irritant poisoning, or are common as the result of the influence of cold, or of acid drinks and unripe fruit. They are also observed as secondary disorders in the exanthemata.

Chronic Diarrhœa.—In chronic diarrhœa the lesions encountered are much more marked than they ever are in the acute form. The mucous membrane is tumid and discolored; its follicles are not infrequently ulcerated. Chronic looseness of the bowels originates in a diarrhœa which is permitted to continue, either from neglect or because the patient remains for a long time exposed to the original cause. The disorder is apt to prove rebellious. When of long standing, the patient becomes gradually weaker and weaker, and more and more emaciated. The abdomen is sunken; the complexion is pale; the eyes are surrounded by a leaden ring. The character of the discharges is various. They are often dark-colored and very offensive. The irritability of the intestines never intermits.

Perhaps the most persistent irritability of the intestines is found in the diarrhœa to which *soldiers* are so liable, and which is apt to pass, no matter what its beginning, into the chronic form of the disease. This complaint, which follows impure water, defective diet, exposure, malaria, and scurvy,[1] which is generally associated with a morbid state of the large as well as of the small intestine, and which combines therefore some of the features of chronic dysentery with those of chronic diarrhœa, is one that often clings to its victim through life: many a soldier, in truth, escapes the bullet and the sword, only to die of the intestinal affection long after his return to his home.

But chronic diarrhœa, as the practitioner of medicine commonly sees it, is often attendant on general constitutional affections, or on abdominal diseases that have led to a secondary disorder of the secretions, or even of the coats of the intestine. Thus, we find chronic looseness of the bowels in scurvy, in pyæmia, in Bright's disease, in scrofula of the mesenteric glands, and in tuberculosis. In the last of these complaints the diarrhœa may be occasioned by changes in the secretions of the intestinal glands; but it is not seldom dependent upon a true *tubercular disease of the intestines*, which, like the disease of the lung, leads to softening and ulceration. The discharges are generally copious and very offensive, and show traces of blood. The diarrhœa is continuous and intractable; the abdomen is retracted, and

[1] Woodward, Outlines of the Chief Camp Diseases, p. 253; see also the elaborate analysis of the alvine fluxes in vol. ii. of the splendid "Medical and Surgical History of the War of the Rebellion," Washington, 1879.

presents spots very tender to the touch. There are marked fever and emaciation, and there may be severe intestinal hemorrhage. Yet, after all, only the signs of tubercle elsewhere furnish any positive indications by which the true nature of the wasting malady can be discerned. Indeed, it may happen that the reverse of diarrhœa occurs; for acute primary miliary tuberculosis may simulate an acute intestinal obstruction.[1] In all cases of suspected tubercular diarrhœa the stools should be examined for tubercle bacilli, and these will be found very generally.

Tubercular ulceration is the most prominent type of ulcerative enteritis. But *ulceration* of the bowel is also met with under other circumstances. We find it in the diarrhœas of children; it occurs then as *follicular ulceration.* Ulceration is also occasionally observed from *cancer*, or as a *solitary ulcer* leading to perforation. The seat of the latter is generally the cæcum or colon. *Albuminuric ulceration*, the careful analysis of Dickinson[2] shows, is almost invariably associated with contracted kidney. *Simple ulcerative colitis* is usually met with in middle-aged persons. It lasts generally about two months,[3] and is ushered in by abdominal pain, which remains a symptom. There is diarrhœa with very thin movements, but there are no dysenteric stools; blood in the discharges is common. The diarrhœa may alternate with attacks of constipation; often there is vomiting. The disease may lead to perforation. *Unhealed typhoid ulcers* form another variety of ulceration of the bowels.

In the diagnosis of all forms of intestinal irritation, we must lay stress on the diarrhœa, on the character of the discharges, on the pain, and on the occurrence of hemorrhage from the bowels. In the discharges, mucus and pus and shreds of tissue are valuable signs. In follicular ulceration little sago-like masses of mucus are met with. The stools may be very frequent; this is especially the case if the ulcer be in the lower part of the colon. Abdominal pain may or may not be associated with tenderness. Pain, as in other forms of colitis, is often referred to the præcordial region. With reference to the frequency of this, Potain[4] tells us that, of one hundred persons complaining of heart disease, about seventy have an affection of the colon.

In the *chronic diarrhœa of strumous children* there is sometimes a

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[1] Thoman, Allg. Wien. Med. Zeit., 1887.

[2] Med.-Chirurg. Trans., vol. lxxvii., 1894.

[3] Hale White, Guy's Hosp. Reports, 3d Series, vol. xxx.

[4] L'Union Médicale, Nov. 1894.

scrofulous infiltration into the intestinal walls, sometimes marked scrofulous enlargement of the mesenteric glands, sometimes both, but in some cases neither. Improper nourishment may be here, as in any other form of the diarrhœa of childhood, the exciting cause of the continued purging.

At times chronic diarrhœa assumes an *intermittent type*, and its malarial nature is clearly proved by the readiness with which the disorder yields to quinine.[1] In this respect malarial diarrhœa differs from a form of diarrhœa we sometimes encounter, in which the pain and discharges come on at an early hour of the day and cease towards evening and during the night.

Another form of looseness of the bowels is the *membranous*. Here the discharges show shreds of membrane, either in connection with the loose stools, or sometimes in such quantities that the whole mass voided seems to consist of them. Griping pains and tenderness usually precede this kind of diarrhœa, which may happen in attacks of a subacute form, or as a persistent and very obstinate disorder: the former variety is the more common. The fecal discharges are loose, but occasionally there is constipation. The disease is often associated with peculiar hysterical symptoms or occurs in neurasthenics. The so-called membranes, in this membranous enteritis, contain a large amount of mucus, as I have elsewhere described.[2]

Dysentery.—Frequent and painful passages of mucus mixed with blood, accompanied by straining and bearing down, are the characteristic symptoms of dysentery. In the acute form we find thirst, restlessness, and fever superadded; and sometimes, especially when the disease prevails epidemically, those symptoms of prostration which are commonly designated as typhoid.

Acute Dysentery.—The acute disorder is at times ushered in by a chill; at times it is preceded by diarrhœa. The fever which attends it is not generally intense. It is the exception to find it exceed 103°, and in light cases the temperature is only slightly raised; the pulse is not tense. More or less pain is always present; it has its seat mostly at some part of the colon, and this is tender on pressure. It is intermitting and shifting, and is often accompanied by a feeling of weight near the anus, which causes a continual desire to go to stool. Yet no relief follows the frequent attempts; the violent straining only adds to the discomfort.

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[1] See contribution by Sanford B. Hunt on *Diarrhœa*, in Medical Memoirs of the U. S. Sanitary Commission, p. 306.

[2] American Journal of the Medical Sciences, Oct. 1871.

The matters voided are small in quantity. They consist of blood mixed with mucus; yet they are composed not simply of mucus, but also of leucocytes, granules, and large quantities of cast-off epithelium, with many swollen, round or ovoid epithelial cells. The stools are in some cases highly offensive, and resemble the washings of meat: in others they are like jelly, or greenish in color. They do not contain fæces, or only here and there small, firm lumps of fecal matter. When the dysenteric inflammation subsides, the bowels are unloaded of their contents; in consequence, the passage of quantities of small, hard masses of fæces is generally a sign that the acute malady is inclining to a favorable termination. Sometimes the stools are very dark and slimy and have a putrid odor, and here and there pieces of sloughed-off tissue can be detected. This kind of stool marks the diphtheritic or gangrenous variety of the malady, though it is not constant even in this.

How long it will take for the disorder to run its course, or whether the acute disease will pass into chronic dysentery, cannot be foretold. Generally this is not its termination; it very often ends, within a week from its beginning, in recovery. But severe cases occur which are of much shorter duration, in which the symptoms hasten on to complete prostration, and death takes place early in the malady. In these frightful cases—mostly epidemic—collapse may happen with almost the same rapidity as it does in malignant cholera.

Dysentery is essentially a disease of hot climates. Eating green fruits, exposure to a chilly night after a hot day, and sleeping on damp ground, are prolific exciting causes. It is occasionally found in combination with malarial fevers, or with scurvy. It also occurs from drinking water full of impure substances or micro-organisms, and is thought to have a bacillus of its own. It may be seen in a sporadic or in an epidemic form. It is very common in armies and in jails. The immediate cause of most of the symptoms is inflammation of the large intestine, and especially of the descending colon. Yet in many cases of dysentery we see phenomena manifested which are clearly not to be accounted for solely by the local morbid appearances, and which show that dysentery mostly belongs to the infectious maladies. In truth, inflammation of the colon may give rise to the symptoms of acute diarrhœa; for it is a great mistake to suppose that the cause of diarrhœa is to be sought only in some abnormal change in the small intestines. Thus, colitis is not always dysentery; and dysentery is often more than mere colitis.

But, whatever be the ultimate cause or the form of dysentery, we find that it presents peculiarities which render it easy of recognition

at the bedside. Yet we must take good care to ascertain that the supposed characteristic tenesmus and bloody discharges are not really owing to *piles*, or to morbid, especially cancerous, growths in the rectum, or to ordinary limited inflammation there. In the latter case, or *proctitis*, there is much pain when the hardened fæces are discharged, the rectum is forced down during the efforts, the sphincter contracts spasmodically. Strangury and hemorrhoids are not uncommon symptoms; and, as the consequence of the inflammation extending to the parts around the anus, an abscess may follow. Rectal pain often extends to the thighs.

Dysentery is not apt to be confounded with *diarrhœa*. This differs essentially from dysentery by the liquid fecal evacuations, and by the fact that neither tenesmus, nor bloody stools, nor discharges of mucus occur. Yet in practice we meet with cases which begin with diarrhœa and terminate in dysentery, or begin with dysenteric symptoms and terminate in diarrhœa, and in which it becomes, therefore, puzzling to say which disorder we are dealing with.

There are some clinical varieties of dysentery which it is important to separate. The ordinary form seen in temperate climates to follow errors in diet or exposure is the catarrhal form. In tropical climates, where dysentery is very common and is met with frequently as an epidemic, we find mostly a kind that is characterized by the presence of the *amœba coli*, or *amœba dysenteriæ*, as Councilman and Lafleur[1] call the micro-organism. *Amœbic dysentery* does not, as a rule, run so rapid a course as ordinary catarrhal dysentery, and local tissue degenerations in the liver, or abscesses of the liver, are common attendants. The abscesses, like the discharges from the bowels, contain amœbæ. The evacuations, as the disease progresses, lose their dysenteric characteristics, except the mucus, and become very liquid; the tenesmus disappears. The amœbæ are most active in alkaline stools. The diarrhœa has marked exacerbations and remissions, and is attended by striking anæmia. The fever is very moderate. In some instances hemorrhage from the bowels, in others peritonitis, happens. It is not unusual in protracted cases for the urine to become albuminous and to contain casts.

In tropical climates, too, though also seen elsewhere in persons who have low forms of pneumonia, or who have become cachectic from scurvy, from Bright's disease, or from long-standing disease of the heart, a form of dysentery attended by extensive exudation and sloughing of the membranes is met with. The *diphtheritic dysentery*,

[1] Johns Hopkins Hospital Reports, vol. ii.

as it is called, has generally high fever, much abdominal pain, great prostration, and delirium. The discharges are very frequent; the blood gradually disappears from them. Vomiting, especially at the onset, is common. In the progress of the case, which is generally to a fatal issue, the temperature becomes irregular, and hiccough is not uncommon.

Chronic Dysentery.—We rarely see chronic dysentery without chronic diarrhœa. At all events, we seldom find instances of the former in which the tenesmus and the discharge of blood and mucus mixed with pus are not accompanied by frequent loose alvine evacuations, by griping, by the same gradual wasting and the same irritability of the bowels as are encountered in chronic diarrhœa; nay, the symptoms of the latter may so obscure the true nature of the malady that what has been regarded as chronic diarrhœa turns out, at the autopsy, to be chronic dysentery. The mucous membrane of the colon is found to be extensively inflamed; its texture altered and irregularly thickened; its surface riddled with ulcers. In such cases the patient goes on steadily losing flesh, and has some elevation of temperature; but no pain on pressure or localized distress exists to denote the ravages the disease is making in the alimentary tube. Many die from exhaustion; others, in consequence of abscess of the liver, which chronic as well as acute dysentery may induce.

Intestinal Hemorrhage, or Melæna.—This is commonly the result of a mechanical hinderance to the flow of blood through the liver, as in cirrhosis, or of disease of the heart, or of a depraved state of the blood,—such as exists in typhus fever, in yellow fever, in scurvy, or in purpura. Occasionally the bleeding proceeds from a fungoid growth in the intestine, or from an ulcer in the duodenum or ileum, or from an invagination, or from fecal impaction, or from an amyloid degeneration of the mucous membrane of the bowel, or is due to a disease of the spleen, or to bursting of an aneurism, or follows extensive burns of the abdominal parietes. In very young infants a discharge of blood, both by the mouth and by the rectum, is not unusual.

The blood passed by stool is generally of dark color, like tar. When it is not, we may infer that it flows from the lower part of the intestine and has not had time to become admixed with other matters. In all such cases, however, we must make sure that it does not proceed from *hemorrhoids*. The exact seat of the hemorrhage cannot be determined; nay, blood may be evacuated by the bowel and not be poured out at all from the intestine, but from the stomach. In some instances the blood accumulates in the bowel, and, before

the clots moulded to its shape are discharged, death results. When the bleeding proceeds from hemorrhoids it is seldom vicarious.[1]

In point of diagnosis the first thing to determine is that what is supposed to be blood is really blood. Very dark bilious stools, or stools blackened by iron, may mislead. If doubt exist, water should be poured on the stool, and, when blood is present, a reddish tinge is imparted to the water; yet more accurate is it to examine with the microscope or the spectroscope.

We next have to ascertain the disease with which the intestinal hemorrhage is associated; and this is often a very difficult matter. We must lay the greatest stress on the history of the case, look for the complaints—of which most have been above mentioned—that are apt to give rise to the bleeding, especially investigating for cirrhosis of the liver; searching for intestinal ulcers in connection with typhoid fever, or tuberculosis, or a duodenal affection; or examining for the evidence of scurvy in the gums and skin; or for purpura with its characteristic spots; or for splenic enlargement, the result of chronic malaria or of amyloid degeneration. Embolism of the superior mesenteric artery may also occasion intestinal hemorrhage. But unless we have with the bloody stools marked abdominal pains, peritoneal exudation, and obvious-causing elements of embolism, or signs of it elsewhere, this diagnosis is most uncertain.

Fatty Diarrhœa.—In some cases in which fatty matter is voided by the bowel, oil is at the same time passed with the urine; in others the urinary secretion is healthy; some cases end fatally, others in recovery; some are found to be connected with a disease of the pancreas, others are not; in some the disorder is not of long continuance, while in others it lasts, with intervals, for years. As a rule, the occurrence of fatty stools is a matter of serious concern. The recognition of the malady is easy. The white, fatty masses, or the oily matter which collects on the discharges, are soluble in ether, and are readily proved to be fat by the microscope. In some instances the bowels are constipated, and lumps of hard fæces are discharged along with the fatty substance. This happened in a marked example of the disorder that came under my observation. The patient, a man of twenty-six years of age, passed a considerable amount of fat, both by the rectum and with the urine. He suffered much from digestive disturbance, from constipation, and from weakness. He had a good

[1] See observations of Cheyne, Dublin Hospital Reports, vol. i., and of Bircombe, Medical Gazette, vol. iv.

[2] Lee on the Rectum.

appetite, but a dislike to fats of any kind. In his case there was, as far as the other symptoms and the physical signs indicated, no tumor in the region of the pancreas. The man's condition was much improved by careful diet and the administration of cinchona and rhubarb; but whether permanently or not I cannot say, as I lost sight of him. I have also met with instances of fatty diarrhœa associated with saccharine diabetes and with disease of the pancreas. In examining into the subject of fatty stools it must be borne in mind that the clay-colored stools of jaundice, owing to the absence of the emulsifying properties of the bile, contain considerable fat, which may be found in oil-drops or as fine needle-shaped fat-crystals.

Diseases attended with Vomiting and Purging.

There is a group of diseases in which vomiting and purging are very prominent symptoms. The most important of these are the various forms of cholera. Now, there are several very different complaints classed together under the head of cholera.

Cholera Infantum.—And first, of the so-called cholera of infants. It is an endemic in the larger cities of the United States during the hot months, and one fraught with danger to all young children. It begins generally with diarrhœa. Vomiting soon follows; and for a time the two go hand in hand; but, unless the case be of short duration, the spontaneous vomiting ceases, or at all events gives way to occasional exacerbations of irritability of the stomach, while the looseness of the bowels remains, or even augments. The discharges are colorless, or yellowish, or greenish. There is thirst; sometimes fever. The abdomen may be sunken or swollen; and it may be tender. Sometimes the disease runs its course within three or four days, at the end of which time the child dies, worn out by the constant vomiting and purging. More generally the disorder is of longer duration; for weeks or for months it continues, the diarrhœa improving and then returning with redoubled severity, and kept up or increased by the irritation of teething. The irritability of the intestinal canal, and the utter impossibility of retaining enough food to nourish the wasting body, gradually wear out the system. The child before death is wan and distressingly emaciated; sometimes hypostatic congestion of the lungs, broncho-pneumonia, boils, suppression of urine, plaintive cries, rolling of the head, strabismus, and coma precede the fatal termination.

Such is a sketch of grave and intractable cases. Yet very many cases are far from being desperate. Under judicious treatment a

large number are annually saved. Recoveries would bear a still higher proportion to the deaths were it not that the greatest sufferers from the disease, the children of the poor, are unable to obtain the means most certain to restore them to health,—change of air. Cooped up in crowded neighborhoods, surrounded on all sides by filth rapidly decomposing under the burning rays of the sun, they are compelled to breathe the hot, noxious atmosphere which, if it do not produce, is certainly a decided agent in keeping up, the complaint.

The disease is an entero-colitis from milk-infection leading to bacterial fermentation in the intestines, with enlargement of the solitary glands, and even at times of Peyer's patches. The researches of Vaughan have demonstrated that a ptomaine appearing in milk, tyrotoxicon, is its most frequent source. Temporary diarrhœas in children occurring in hot weather could alone be mistaken for the disorder. But the fact that they are temporary, not followed by vomiting, and not associated with the grave symptoms of approaching collapse, shows us the difference.

Cholera Morbus.—This, or *cholera nostras*, is, like cholera infantum, a disease of the hot season; yet it is also observed at other times of the year. But, although the chief predisposing cause is undoubtedly heat, there is generally an exciting cause which develops the disorder,—such as exposure, checked perspiration, drinking large quanties of ice-water, or imprudence in eating. The attack is characterized by spasmodic pains in the abdomen, by cramps in the legs, by rapid loss of strength, and by repeated vomiting and purging. The matter ejected both from the stomach and from the intestines is liquid, and contains a large quantity of bile. In truth, the affection is in reality a cholera, a flow of bile, which its more formidable namesake, Asiatic cholera, is not. Finkler and Prior have found in the stools a comma bacillus, *vibrio proteus*, which is larger and thicker than the bacillus of Asiatic cholera, but with shorter spirilla, and cultures of which, unlike the latter, rapidly liquefy in gelatin, and grow on potato even at ordinary temperatures.

Cholera morbus may be preceded by colicky pains, nausea, and rumbling in the intestines. More generally it comes on suddenly. When at its height, the cramps in the calves of the legs cause the muscles to rise up in hard, knotty masses; the stools are fetid; the vomiting is constant; the thirst is great, and the skin is cool or cold. But the patient does not remain long in this condition. In the course of a few hours, or at the utmost of a day, the symptoms mitigate, or yield entirely to treatment; and, pale and visibly emaciated though he be, he speedily regains his health. Only in some cases the disease

proves intractable, and, after running on for several days, passes into a state of hopeless collapse.

There are not many morbid states with which cholera morbus is likely to be confounded. It may be mistaken, as we shall presently see, for epidemic cholera. We find many points of similarity between it and irritant poison; but there are also strong points of difference. The vomiting and purging produced by an irritant poison do not come on at the same time: the vomiting precedes the purging, and there may be bloody evacuations. The pain is first in the epigastrium, thence it may spread. Moreover, we often detect signs in the mouth or fauces which prove the irritating character of the substance swallowed. The vomiting and the subsequent acute gastritis are accompanied by fever, which is not the case in cholera morbus.

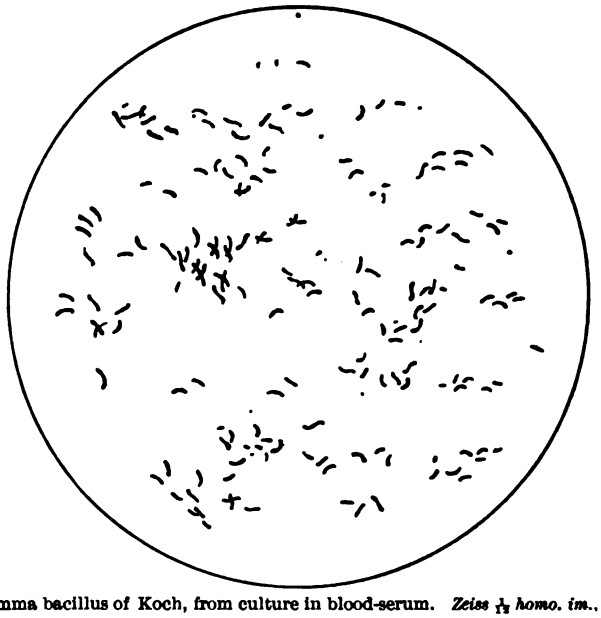
Cholera.—The formidable complaint known as epidemic cholera, Asiatic cholera, malignant cholera, or by the simple name of cholera, has some striking features of resemblance to the disorder just considered. It shares with cholera morbus the vomiting and purging, the cramps, the sudden depression; but it is an affection of different origin and of much more serious import, and presents symptoms not encountered in the cholera that occurs yearly during the hot weather. And although, on account of the gastric and intestinal disturbances which form so prominent a part of its manifestations, it is here described among the disorders of the alimentary tube, I am doing so for the sake of clinical convenience, and contrary to sound pathology; for cholera is not an affection either of the stomach or of the intestines; it is an epidemic constitutional disorder of the most formidable character generated by a poison transmitted to us from the East. The poison leads to a casting off of the epithelium of the mucous membrane of the alimentary tube; perhaps to changes in the membrane. But the engorged veins all over the body; the exosmosis of the watery parts of the blood; the frightfully rapid prostration; the sudden blight which befalls the nervous powers,—are elements which are even more characteristic.

The access of cholera is at times sudden and most unexpected; the patient, previously in good health, is stricken down without warning by the force of the poison. More generally there is a premonitory stage: a stage of languor, low spirits, uneasiness, headache, and diarrhœa. The effects of the morbific matter are indeed visible in hundreds of individuals who, during the prevalence of cholera, suffer from these premonitory symptoms without any of greater danger arising. Nay, the same influences which give rise to *choleraic diarrhœa* in healthy persons have the effect of rendering the

bowels of those habitually constipated regular, and sometimes even loose.

When the malignant disease is fairly developed, there is vomiting as well as purging. The contents of the stomach and intestines are first voided, and then large quantities of a rather turbid fluid resembling rice-water, with whitish particles like rice floating in it. They are the epithelial cells of the alimentary tube, which have been thrown off from the mucous membrane; and in the dejecta we find the comma bacillus discovered by Koch. This may be seen by examining microscopically the bacilli obtained from a small amount

FIG. 55.



The comma bacillus of Koch, from culture in blood-serum. *Zeiss* $\frac{1}{18}$ *homo. im.*, Oc. 4.

of cholera dejection that has been mixed with an equal amount of alkaline meat broth at a temperature of 30° to 40° C. and allowed to stand for twelve hours in an open glass. The cholera bacilli develop on the surface. They are readily stained, in about ten minutes, with a diluted alcoholic solution of fuchsin or methyl violet. They are decolorized by Gram's process. After the staining, which must take place with the infected side downward, the cover-glasses are washed in water, dried with the prepared side uppermost, and mounted in Canada balsam. Prior to the staining a drop of the infected broth or a particle from a stool is dried in air, after having been rubbed between two cover-glasses and passed three times through the flame of a Bun-

sen burner. The bacilli of cholera may be recognized even without the microscope by a rose-violet color, the *cholera reaction*, that becomes apparent in a few minutes if a ten per cent. hydrochloric acid solution is added to cholera cultures. The cholera bacillus is confined to the intestine. In the extensive observations made by Shakespeare in India and elsewhere[1] it was not detected in the blood or in the tissues or organs outside of the intestinal canal. The cholera toxine derived from the bacilli has been specially studied by Pfeiffer.

Simultaneously with the vomiting and purging, or very shortly after, come on severe spasmodic pains in the abdomen, and cramps of the muscles of the belly and of the extremities. With all this there are a burning sensation in the epigastric region; an unquenchable desire for cold drinks; a cool skin; a pulse slightly more frequent than normal; a temperature which may be normal or may fall to about 95° F.; oppressed breathing; and rapidly progressing exhaustion. The case now stands on the verge of collapse. Should this follow, the pulse becomes hardly perceptible. The discharges cease, and so do often the cramps. The skin is cold, covered with a clammy sweat, and has a bluish look. The nails and the lips have the same unnatural appearance. The whole body shrinks, and seems at times almost to wither visibly even while under inspection. The countenance assumes the aspect of death; the eyes are sunken and have a glassy look. The temperature is low, it may fall below 90°; but while very low in the mouth or axilla, it may be 103° or more in the rectum. The intellect is commonly clear; but, when the patient talks, the words fall strangely on the ear. It seems as if a corpse had spoken, and the voice is husky and faint. The tongue and the expired air are cold. No symptom, indeed, has struck me more forcibly than the icy breath.

But the symptoms do not always take place in the order described, nor are they all uniformly present. The vomiting and purging may be wanting from the onset, and so too may the cramps. Only one symptom is never absent,—the tendency to early sinking. Sometimes a stage of perfect collapse is reached with frightful rapidity: instead, as is commonly the case, of several hours elapsing before complete prostration comes on, the vital powers are at once laid low by the assault of the dreadful malady. When cholera last prevailed in Philadelphia, I attended a woman who, at six o'clock in the morning, was in perfect health, and who, in a little more than half an hour afterwards, was lifeless. There was neither vomiting nor purging;

[1] Report on Cholera in Europe and Asia, Washington, 1890.

nothing but cramps, stupor, and speedy collapse. Such cases are not uncommon in the home of cholera,—India. Post-mortem inspection shows the thin rice-water fluid locked up in the alimentary canal. Nature makes an effort to eliminate the poison; but before she completes her task, life is palsied.

In those cases that recover, or in those of light character, *cholerine*, the vomiting and purging gradually subside, the skin becomes warm, the pulse fuller, the abdominal pain ceases, the urine—which, while the disease is at its height, is not passed, perhaps not secreted—is again voided, the patient falls into a refreshing sleep, and, the symptom most favorable of all, bile reappears in the stools. Even in apparently hopeless cases of collapse we may be fortunate enough to witness these favorable changes. But, where the prostration has been great, the reaction is apt to be violent. A decided fever of low type, with rapid pulse and heat of skin, and attended very often by alarming cerebral symptoms, succeeds; and the urinary secretion, even if it had been restored, becomes again very scanty. Thus the period of reaction brings with it new dangers, and of a kind which are sometimes insurmountable. And this low form of fever, very similar to typhoid, though readily enough distinguished by the preceding symptoms, may last for upward of a week before death takes place or the signs of danger gradually yield. Now, this *cholera typhoid* may be preceded by scanty urine and marked uræmia, but it may also exist independently of this morbid state, though probably also due to the blood being loaded with broken-down material. In cases in which uræmia sets in, whether it be followed or not by a fever of low type, there is at first but little, if any, heat of skin, and a slow pulse; the patient is wild, restless, or drowsy; the kidneys act very imperfectly, the urine is greatly deficient in urea, and usually contains albumin. These are very dangerous cases, and if the secretion be seriously retarded for more than twenty-four hours they are likely to perish. Other complications that may arise are pneumonia, pleurisy, suppurative parotitis, and protracted nephritis.

In any case of cholera, convalescence is apt to be slow. For weeks or months irritability of the intestinal canal remains; and I have met with instances in which it has never disappeared. In convalescence, too, we may find constantly recurring cramps in the arms and legs.

It would be needless to go into any minute description of the differences between cholera and other affections; its features are not to be mistaken. *Cholera morbus* is the only disorder which really resembles it. The dividing-line is drawn by the absence of bile in the

discharges, the rice-water evacuations, the greater severity and more rapid progress of the symptoms, the bluish color of the surface in the stage of collapse, and the epidemic character of the more fatal disease. In the presence of the cholera bacillus in the evacuations, and in the speedy collapse, lie, even in doubtful cases, the proofs that we are dealing with malignant cholera; for sometimes rice-water discharges occur in bad cases of cholera morbus; occasionally, too, this disorder appears to be epidemic; but it is only so on a very small scale. To speak more accurately, it is an endemic on a large scale.

The mortality of cholera is very various. In many epidemics one-half, or more than one-half, die. In some the havoc is far less. The first cases that occur almost invariably perish; and, taken altogether, the malady ranks among the most destructive to life. Its epidemic visitations are what the plague was to the Europeans of the seventeenth century, and what yellow fever still is to the inhabitants of this continent.

SECTION III.

DISEASES OF THE LIVER.

The physical characteristics of disease of the liver have been already discussed. Let us now look at some of the symptoms.

Pain is one of these. It is generally dull, and radiates from the seat of the liver to the upper portion of the thorax, to the scapula, to the shoulder, and to the umbilicus. Commonly it is persistent and increased by strong pressure. As happens with other symptoms of disease of the liver, with vomiting, with jaundice, it may be noticed that the pain is sometimes strangely periodical, suggesting malaria, but uninfluenced by quinine.[1] *Digestive troubles* are usual accompaniments of hepatic affections. They are of all grades, from mere indigestion to the signs announcing chronic gastritis. *Disturbance of the portal circulation* is another frequent consequence of disease of the liver. The flow of blood is interfered with, and the result is seen in the occurrence of dropsy, of piles, of partial peritoneal inflammation, of hemorrhages from the engorged stomach and intestines, and of enlargement of the spleen and of the veins on the surface of the abdomen.

Jaundice.—The most significant manifestation of hepatic disorder is jaundice. This marked sign shows itself by the yellow tinge imparted to the skin and to the conjunctiva. Besides, icterus is usually

[1] See on this subject a paper by Cyr, Arch. Gén. de Méd., May, 1883.

attended with depression of spirits; with slow pulse; with itching of the skin; with high-colored urine, in which the main ingredients of bile can be detected, and sometimes small quantities of albumin, or hyaline and epithelial casts without albumin; with constipation, the fæces passed being hard and knotty, and often of bad odor, and almost devoid of color, or of a leaden hue.

Jaundice is due to the presence of biliary constituents in the blood; they get there from the bile, in consequence of some impediment to its outward passage, being reabsorbed and conveyed into the circulation; or it happens because the liver-cells cannot perform their functions; or because some poison changes the proper relation between blood-destruction and cell-action in the liver; or the bile pigments may be formed directly from hæmoglobin without the agency of the liver-cells; for this, too, is a view of toxæmic jaundice with blood-destruction that seems best to apply to certain cases.

The *diagnosis* of jaundice is easy. The only morbid signs with which it is liable to be confounded are the slightly yellowish hue of chlorosis, or of some cachectic conditions combined with organic visceral disease, and the yellow appearance of the conjunctiva which is natural to some persons. The changed color of the countenance due to chlorosis is told by its association with a bluish-white or pearly-tinted eye, and with pale lips and tongue and transparent ear. The absence of a yellow tint from the conjunctiva is of equal importance in discriminating from jaundice the yellowish hue of cancer, of malaria, of lead poisoning, and of granular kidneys. The history of the case also aids us. The yellow look of the eye sometimes found in health, and at times dependent on subconjunctival fat, is known by the unequal distribution of the color and by the absence of a yellow hue of the complexion. But in negroes—and it is in them especially that we meet with the discolored conjunctiva—we have to judge by the character of the coloration alone. In any doubtful case, the chemical tests for bile-pigment in the urine will solve the doubt. Yet there is a form of jaundice, the so-called *acholuric jaundice*, in which neither bile-pigment nor urobilin is found in the urine, but in which a yellowish discoloration of the skin is very marked, and urobilin and other biliary pigments are present in the serum of the blood. It is a chronic disorder, occurring in neurasthenic and in dyspeptic persons, especially in those with hyperacidity.[1] The conjunctiva has only a very slightly yellowish tinge.

When once jaundice has been recognized, the difficulty in diagnosis

[1] Hayem, Bull. et Mém. de la Soc. Méd. des Hôp. de Paris, May, 1897.

may be said to begin. Of the many distinct sources of icterus, which one is before us? Now, clinically speaking, the causes may be thus grouped: 1. Diseases of the liver. 2. Diseases of the bile-ducts. 3. Diseases of parts remote from the liver, or general diseases leading to a disorder of the viscus. 4. Certain poisons acting upon the blood. In the first two of these causes there is, as it were, a mechanical difficulty impeding or arresting the excretion of bile; in the third and fourth no impediment exists.

1. The jaundice connected with diseases of the liver is, as a rule, recognized by its association with changed dimensions of the organ, and with pain or other palpable signs referred to the hepatic region. It is met with in all disorders of the liver, but does not exist in all in the same degree of intensity. It reaches a high development and is combined with brain symptoms in acute yellow atrophy. In fatty liver, in waxy liver, in cancer, in cirrhosis, and in acute hepatitis, it is not marked, and may be, indeed, absent: in truth, it can hardly be looked upon as belonging to the first-mentioned morbid states. The jaundice of this class of cases is due to interference with the secreting function of the liver-cells.

2. Jaundice arising from disease of the larger biliary ducts, such as their catarrhal swelling; or in consequence of their obstruction by pressure exercised by a morbid enlargement of the adjacent parts, as of the pyloric extremity of the stomach or the pancreas; or by tumors, aneurismal, cancerous, or fecal, closing the orifice of the duct; or by tumors of the gall-bladder and bile-ducts; or by the stoppage of the ducts by inspissated bile or a biliary calculus, or by hydatids or foreign bodies from the intestines,—is a form of the malady in which the icterus is commonly intense. The obstructive jaundice occasions no head symptoms; and when these are absent in a case of very deep jaundice, when, further, the stools are completely discolored, we are generally correct in attributing the morbid phenomena to an impediment to the flow of bile through the common bile-duct or the hepatic duct.

In the jaundice due to reabsorption—precisely the form of jaundice, therefore, that happens if any serious obstacle in the biliary passages exist—the biliary acids pass into the blood, and thence into the urine. But this is not a certain sign of obstructive jaundice; for in the other forms of jaundice, as in the non-obstructive, they may be present, though in lesser amounts, and traces of the bile-acids may be found even in healthy urine.

3. Illustrations of jaundice following some local lesion of other parts of the body, or appearing in the course of an infective disease,

are furnished by the jaundice which happens in some cases of pneumonia, or in peritonitis, or which is encountered in pyæmia, in remittent, in typhus, in relapsing, or in yellow fever. In these fevers the yellow hue is generally found to be connected with structural changes in the organ. But, besides the interference with the secreting action of the cells, the blood alterations in non-obstructive jaundice must be considered; there is certainly increased corpuscular destruction. But the blood-change may, the observations of Afanassiew and others prove, lead to increased viscidity of the bile, and compression of the bile capillaries; thus the jaundice is really in part obstructive.

To recognize the form of jaundice under discussion, we must examine all the viscera of the body with care, laying stress upon the history of the case and the phenomena attending the jaundice.

4. Poisons acting upon the blood sometimes give rise to jaundice very rapidly; for instance, the jaundice from snake-bites or from pyæmic affection is apt to be suddenly developed. As a rule, the tint is light. In the history of the accident and the signs of alteration of the blood we possess the means of distinguishing this form of jaundice. Certain mineral poisons, such as phosphorus, copper, antimony, come into the same category. Chloroform and ether, too, lead to abnormal blood-changes producing jaundice. The deep jaundice of arsenuretted hydrogen and of toluyindianin is largely obstructive, caused by the irritant action of products in the bile. As a general fact it may be stated that in all these kinds of toxæmic jaundice, the icterus is apt to be light, but the constitutional symptoms are severe; bile is not wholly absent from the stools.

The urine enables us to a certain extent to tell blood jaundice from jaundice caused by liver disorder. We find, besides an excess of urobilin, hæmoglobin in the urine, or get from its hæmatin the hæmin crystals of Teichmann. These are obtained by drying urine on a slide, adding a little salt, and then glacial acetic acid under the cover-glass. The slide is heated until bubbles rise, and on cooling the characteristic blood-crystals form.

Thus, then, we can bring, clinically speaking, most of the varieties of jaundice under one or the other of the four heads mentioned; and, roughly speaking, they come really under two,—obstructive jaundice, where the disorder results from obstruction of the common duct, and jaundice without such obstruction. But there are a few kinds of jaundice which it is not easy to classify with precision: one of these is the jaundice from mental emotion.

As regards this, no satisfactory explanation has been given. All we know is, that violent anger or fright may lead within a very brief

space of time to the development of jaundice, and that the quickly occurring discoloration is not dangerous or of long duration. The perverted innervation caused by concussion of the brain leads to a similar kind of jaundice as that from emotion. It is thought by some that a spasm of the bile-ducts obstructs the flow of bile; by others that the hæmoglobin of the blood, instead of breaking up into normal bile pigment, may break up into abnormal pigment, and that the icterus is really a urobilin jaundice, which gives rise to the icteric skin and conjunctiva.

If icterus last upward of two months it is always a matter of some danger, as showing, in all likelihood, an organic lesion of the liver or of the biliary passages, or unyielding pressure on them. Unfavorable, too, is it if the discoloration of the skin be attended with cerebral symptoms, or accompany affections of the blood, or be associated with wide-spread ecchymoses, or a very dark color of the skin. Indeed, cases of "green" or "black" jaundice generally prove fatal.

Before examining the hepatic maladies according to their clinical features, let us look at their pathological classification:

DISEASES OF THE LIVER.

Diseases of hepatic parenchyma.	Hyperæmia	Acute congestion.
		Chronic congestion.
	Inflammation and its consequences	Acute hepatitis.
		Chronic hepatitis.
		Interstitial inflammation; cirrhosis, atrophic and hypertrophic.
		Abscess.
		Softening.
		Syphilitic hepatitis.
	Atrophy	Acute yellow atrophy.
		Simple chronic atrophy.
		Red atrophy.
	Hypertrophy	Partial.
		General.
	Degeneration and new formations	Fatty liver.
		Waxy liver.
		Pigment liver.
		Cancer.
		Sarcoma.
		Lymphatic growths.
		Gummata.
		Tubercle.
		Hydatids.
		Simple cysts.

DISEASES OF THE LIVER.—*Continued.*

Diseases of biliary passages.	Inflammation of gall-bladder (cholecystitis) and gall-ducts (cholangitis)	Catarrhal. Exudative. Suppurative.
	Occlusion of biliary passages.	
	Dilatation of gall-bladder.	
	Morbid growths.	
	Foreign bodies; concretions, such as gall-stones.	
	Biliary fistulæ.	
Diseases of blood-vessels.	Of hepatic artery	Inflammation. Sclerosis. Aneurism.
	Of hepatic vein.	
	Of portal vein	Suppurative inflammation. Thrombosis.

Acute Diseases of the Liver attended generally with Slight Enlargement of the Organ, and with more or less, though rarely much, Jaundice.

Acute Congestion.—This arises from organic disease of the heart, from obstructed portal circulation, from irritating food and drink and disturbed digestion, from gastric or intestinal catarrh, or from malarial poison; sometimes it is caused by a high temperature, by a blow on the hepatic region, by arrest of the menstrual flow, by a protracted chill, by violent exercise, or, as Frerichs points out, by injury to the semilunar ganglia. The acute congestion is characterized by pain in the right shoulder and loin, by an unpleasant sensation of weight and of tension in the right hypochondrium, increased after meals, and by nausea and vomiting. At the same time the action of the bowels is deranged, being generally too frequent; the tongue is coated; there is flatulency, as well as depression of spirits, with loss of appetite and of strength; and the liver is somewhat enlarged. But we find ordinarily only slight jaundice, and no fever. Gradually these signs disappear; the increased hepatic dulness, however, remaining for some time after the gastric and intestinal disturbances have abated. These always bear a marked relation to congestion of the liver, both as cause and as effect. The acute disorder may gradually pass into a chronic hyperæmia.

Acute Hepatitis.—The symptoms of this affection are much the same as those of acute congestion, except that we observe rise of temperature, and in some cases enlargement of the spleen, and albumin in the urine. The pain is dull, and is increased on pressure, yet

not much so, unless the peritoneal covering of the liver be involved. But acute hepatitis is not a well-defined affection, and we know little of it except in connection with dysentery, particularly with amœbic dysentery. In hot climates it often terminates in suppuration, and pus collects in the substance of the liver. The occurrence of this, the *tropical abscess*, as Murchison[1] calls it, is indicated by recurring rigors, by fever of remittent type, by clammy perspirations, by prostration and loss of flesh. Not infrequently, too, a decided fulness of the side may be noticed, and occasionally careful palpation detects deep-seated fluctuation. After an abscess has formed, the danger is great; secondary abscess may follow, and the patient is apt to perish from peritonitis, or from blood poisoning. Yet recovery may take place. The matter may be discharged through the abdominal walls, or burst into the intestine, or find its way through the diaphragm into the pleural cavity, to be discharged through the lung. But, as the phenomena of abscess of the liver following acute inflammation are in the main the same as when the suppurative hepatitis is consequent upon other morbid states, we shall not here consider what we shall presently fully examine. The pyæmic liver abscess is the one of greatest similarity.

The maladies resembling acute congestion or acute hepatitis are:

PERIHEPATITIS;

INFLAMMATION OF THE PORTAL VEINS;

PIGMENT LIVER;

CHRONIC HEPATIC DISEASES WITH ACUTE SYMPTOMS;

ACUTE NON-HEPATIC DISEASES WITH JAUNDICE;

DIAPHRAGMATIC PLEURISY;

ACUTE INFECTIOUS JAUNDICE;

INFLAMMATION OF THE BILIARY PASSAGES;

ACUTE YELLOW ATROPHY.

Perihepatitis.—Inflammation limited to the serous covering of the liver is not a frequent disease. Unless it be of syphilitic origin, it is scarcely ever observed as a primary affection; it is generally caused by the extension of inflammation from parts adjacent to the liver,—as from the stomach, intestines, diaphragm, or pleura,—or of a chronic peritonitis; or it is an attendant upon disease of the liver itself. In the latter case it presents no peculiar symptoms, except that it adds tenderness to the signs of the hepatic malady it complicates. Its most marked signs are, besides the decided tenderness, severe pain upon motion or deep inspiration, and marked increase of the pain when the patient lies on either side; an occasional grating friction sound; and a

[1] Diseases of the Liver, 2d edit., 1877.

normal or increased size of the gland. The history of the case, especially its association with interstitial nephritis, chronic peritonitis or ascites, tenderness over the spleen from coexisting inflammation of its capsule absence of jaundice, and slight fever are also signs of value. The smaller size of the liver, the absence of tenderness localized over it, and the rapidly forming and, after tapping, quickly recurring dropsy, distinguish cirrhosis of the liver with peritoneal involvement from perihepatitis. The latter affection, certainly the chronic hyperplastic form, has generally an acute beginning and runs a slow course; the ascites often becomes stationary.[1]

Inflammation of the Portal Veins; Pylephlebitis.—An inflammation of the portal veins, terminating in suppuration or their infection by a general pyæmia, or through local processes in the portal circle, is very liable to be mistaken for suppurative hepatitis. Nor are there, in truth, any positive symptoms by which we can discriminate between the two maladies. Still, we may suspect that the veins, rather than the structure of the liver, are the seat of inflammation, if, with the signs of acute and painful enlargement of the organ, we find jaundice, thin and copious stools, irregular fever and profuse sweats, occasional chills, emaciation, increase in the size of the spleen, typhoid symptoms, without apparent fluctuation or other signs of an hepatic abscess; if there exist pains in the epigastrium or right hypochondrium, or shooting to the lumbar and sacral regions; if following these symptoms appear swelling of the veins of the abdominal walls and striking evidences of hectic fever or of peritonitis; and if these phenomena be encountered in a person who, on account of a previous affection of the intestines or the appendix or the spleen, or of any other organ having a connection with the portal circulation, is liable to disease of the portal system. Marked enlargement of the spleen is a constant feature of impediment in the portal vein, whether from inflammation or from thrombosis.

Pigment Liver.—In accumulation of pigment in the liver, which is most common as the result of a deep malarial poisoning, the liver is not the only organ implicated in the morbid process: the spleen is commonly affected; the blood becomes anæmic, contains the malarial corpuscles and large quantities of pigment, and pigment accumulates in the kidneys or in the brain. Now, the effect of all this is to occasion marked symptoms, besides those referable to the derangement of the liver; for it is not unusual to find grave cerebral disturbance, albuminuria, hemorrhage from the intestines, profuse diarrhœa, and

[1] Schmaly and Webber, Deutsche Med. Wochenschrift, 1899, No. 12.

enlargement of the spleen. The fever that accompanies the morbid condition is apt to be of an intermittent type; the jaundice is generally slight. In India, pigmentary degeneration of the liver tends to suppurative hepatitis.[1]

Chronic Hepatic Diseases with Acute Symptoms.—We occasionally meet with patients who seem to be laboring under an acute affection of the liver, either some form of inflammation of the liver-structure or of the biliary passages, or congestion of the liver, but in whom the acute symptoms have merely supervened upon a chronic complaint. Such cases are puzzling; we may have to wait for their solution until the acute symptoms subside. In hepatic cancer the sudden and rapid development of the malady amid the signs of acute congestion is not very uncommon. Occasionally the peculiar physical phenomena of individual hepatic diseases, such as the nodular tumors of a malignant growth, or the fluctuation of a hydatid cyst, will assist materially in the diagnosis.

Acute Non-Hepatic Diseases with Jaundice.—There are many acute affections, such as pneumonia, pyæmia, puerperal fever, and some forms of sepsis, in which jaundice may coincide with febrile symptoms and excite suspicions of hepatitis. But the yellowness of the skin which may attend the non-hepatic disorders mentioned is accompanied by symptoms so different that a mistake is not likely to arise if the history of the case be taken into account and other viscera besides the liver be explored.

Diaphragmatic Pleurisy.—Inflammation of the pleural covering of the diaphragm may give rise to symptoms that point to an acute affection of the liver. We find pain in the right hypochondrium, nausea and vomiting, dry cough, and embarrassed respiration. But the pain in diaphragmatic pleurisy is far greater than even in perihepatitis, is more suddenly developed, and is much more aggravated by movements and by full inspiration. The diaphragm on one side is immovable; the hypochondriac region is retracted; the breathing is purely costal and short; the difficulty in breathing amounts to orthopnœa; the body is bent forward. We often encounter hiccough, great anxiety, sometimes delirium, attacks like angina, a sardonic grin on the features, a cough that comes on in frequent paroxysms; and although, as a case recorded by Andral[2] proves, there may be jaundice, yet this is in reality so generally wanting as scarcely to belong to the symptoms of diaphragmatic pleurisy. Then in this complaint we perceive friction sounds,—though the physical signs will not always aid

[1] Aitken's Practice of Medicine, vol. ii.

[2] Clinique Médicale, tome ii.

us, being often uncertain, mostly out of all proportion to the gravity of the general symptoms, and consisting simply in enfeebled breathing, with perhaps a few fine moist râles at the lower portion of one side of the chest. Fever may be slight or marked; it is generally ushered in by a chill. There is usually, in addition to the pain along the cartilages of the false ribs, which is readily evoked by pressure, a tender spot in the epigastrium, on a level with the tenth rib, one or two finger-breadths from the linea alba. There are shooting pains along the clavicle and in the tract of the superficial cervical plexus, and the phrenic nerve of the affected side, pressed on in the neck, is very sensitive. The pain on pressure is most intense along the costal insertions of the diaphragm, especially of the tenth rib; it is stated that upward pressure affords a means of diagnosis, as it relieves the pleuritic pain.[1] The difficulty in expectorating, owing to the pain, may be so great as to hasten death.[2]

Acute Infectious Jaundice.—This malady,[3] also known as *Weil's disease*, presents symptoms of an acute hepatitis. But it is probably not a disease of the liver at all, but rather an infectious fever due to the invasion of a specific micro-organism through the gastro-intestinal tract. Jaeger[4] has, in cases of Weil's disease, isolated from the urine during life and from the tissues after death a short curved rod, provided with cilia, which he designates "bacillus proteus flavescens." Weil's disease is marked by jaundice, swelling of the spleen, nephritis, and blood-alteration. It mostly affects vigorous young men in hot weather; butchers and soldiers are especially liable to it. It has been also observed in persons who have bathed in water contaminated by fowls suffering from an analogous disorder,[5] and in epidemics. It begins abruptly with headache, dizziness, and decided elevation of temperature. The jaundice is, as a rule, moderate, the liver slightly swollen and painful; there is great weakness, with delirium and somnolency, increased thirst, and general malaise, with loss of appetite. Besides albumin and tube-casts, the urine may contain blood; both bile-pigment and bile-acids are found in it. There are pains in the limbs, especially in the calves; the bowels are usually loose. The symptoms abate quickly; from the seventh to the eighth day the temperature falls gradually to normal, but the fever may last from ten to

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[1] British Medical Journal, Aug. 1871.

[2] Frank Donaldson, Jr., Amer. Journ. Med. Sci., April, 1885.

[3] Described by Weil, Deutsches Archiv für klin. Med., Bd. xxxix.

[4] Zeitschrift für Hygiene und Infektionskrankheiten, Dec. 9, 1892.

[5] Jaeger, *loc. cit.*

fourteen days. A return of fever after a period of its absence from one to seven days may happen, but this return does not last more than three to six days. The convalescence is extremely slow. Fatal cases have presented fatty degeneration of the liver, acute parenchymatous nephritis, and enlargement of the spleen.[1] The disease resembles *relapsing fever*, but the spirilla have not been found in the blood. Nor is defervescence attended with a critical discharge followed by subnormal temperature. Further, the ascent of the temperature of the secondary fever is gradual, while that of the paroxysm of relapsing fever is sudden. The return of the fever makes it unlike *abortive typhoid* with bilious symptoms. Then it shows no eruption, except herpes and an erythema.[2] Besides, jaundice and urine containing blood are rare in typhoid fever.

Between *acute yellow atrophy* of the liver and Weil's disease there is a close resemblance. But the former has a prodromal period, while the onset of the latter is abrupt. The second is attended with elevation of temperature of peculiar range; in the first the temperature is, as a rule, not elevated, and may be subnormal, and the bowels are constipated. In acute yellow atrophy the jaundice is gradually progressive and the liver is at first enlarged and subsequently reduced in size; the jaundice of Weil's disease is slight and soon subsides, and the liver remains enlarged throughout the attack. In acute yellow atrophy the urine may contain albumin and tube-casts, but there are not the pronounced symptoms of nephritis that Weil's disease presents. The tendency to hemorrhages is far greater in acute atrophy of the liver than in infectious jaundice. The one condition is almost invariably fatal; the other is, as a rule, followed by rapid improvement and recovery. Weil's disease in some respects resembles yellow fever, but it is an affection of several paroxysms.

Inflammation of the Gall-Bladder and Gall-Ducts.—The symptoms of this vary materially according to the parts specially affected, as well as to the kind of inflammation, whether suppurative or not. When the gall-bladder alone is inflamed, we have *cholecystitis;* when the bile-ducts alone, especially the finer ducts, *cholangitis*, which is generally infective or suppurative. The most common form of inflammation by far is inflammation of the ductus choledochus, chiefly at its terminal portion, and catarrhal.

Catarrhal Jaundice.—The morbid process is nearly always propagated from the stomach or intestines, and nausea, furred tongue, a

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[1] Jaeger, *loc. cit.*

[2] Fiedler, Deutsches Archiv f. klin. Med., Feb. 1888.

feeling of weight in the epigastrium, feverishness, and diarrhœa occur previously to the discoloration of the fæces, to the jaundice, to the increased hepatic dulness, and to the slight tenderness on pressure in the right hypochondrium: in other words, the symptoms of gastric or gastro-intestinal catarrh precede those of "icterus catarrhalis."

Catarrhal icterus does not cause any great enlargement of the liver, and the slightly swollen organ remains smooth on palpation. Nor is the tenderness decided, except over the tumid and projecting gall-bladder. The jaundice, at first slight, becomes after a few days, as the duct is obstructed, intense, and the stools are white and devoid of bile. There is now no fever, or this is but very slight: the pulse is usually slow. The affection is the most common cause of marked jaundice in young persons: when found in the middle-aged or in the old it is apt to be associated with a gouty diathesis or to have followed syphilis: and at any age it may be secondary to other diseases of the liver, or to gall-stones, and is then apt to be more lasting.

Generally catarrhal icterus is a tractable disorder, and after continuing for two or three weeks, it usually subsides. But it may persist for as many months: and in rare instances the inflammation leads to an occlusion of the common duct, and to a fatal issue. I had such a case in 1863 under my charge at the Philadelphia Hospital. The patient, a man upward of sixty years of age, died deeply jaundiced and comatose. He had presented, during life, the signs of enlargement of the liver; little or no tenderness in the hepatic region: no fever: but much gastric irritability and obstinate constipation, both of which had existed for three weeks prior to a noticeable discoloration of the skin. The disease was, as far as could be ascertained, of only two months' duration: and the jaundice steadily deepened from the time of its first appearance. At the autopsy, the gall-bladder was found enormously distended, its coats thin, yet otherwise scarcely abnormal: but the common duct was obliterated by inflammation. The stomach and the upper bowel were congested, while the coats of the stomach towards the pylorus were thickened. A similar case has been described by Tyson.[1]

Now, it is not generally difficult to distinguish catarrhal jaundice, except in those very exceptional cases in which the common duct or the hepatic duct is obliterated. It differs from *congestion of the liver* by the different etiological elements,—the one disorder happening commonly in connection with disease of the heart, or an obstruction of the portal circulation, or a miasmatic poison: the other following usually exposure

[1] Transactions of the Pathological Society of Philadelphia, vol. iv.

to cold and damp, or the eating of quantities of indigestible food. Then, inflammation of the gall-ducts gives rise to decided jaundice.

Catarrhal jaundice may occur as an accompaniment of some general morbid condition, or in an epidemic form. The epidemic cases are distinguished by the history, by the tendency to acute disease of other organs, such as the lungs and kidneys, by pain in the region of the liver, and by enlargement of the spleen.[1]

From the jaundice of chronic hepatic maladies—such as *cancer* or *cirrhosis*—we separate catarrhal icterus by the non-existence of the physical signs of these maladies, by its acute course, and by the dissimilar progress of the symptoms. Still, as regards cancer we must bear in mind that we encounter in elderly gouty persons cases of long-persisting catarrhal icterus attended with frequent vomiting and marked emaciation which strongly resemble cancer, yet slowly yield to treatment. Inflammation of the biliary passages with the jaundice arising in consequence of *biliary calculi* is distinguished by the severe pain, the sudden appearance of the icterus subsequent to the paroxysms of pain, its increase after them, and its often rapid fading after the gall-stone is voided.

In some cases of inflammation of the biliary ducts, especially where an occlusion of the ducts takes place, a peculiar paroxysmal fever is developed, with temperature ranging from 103° to 105°, which is readily mistaken for a malarial outbreak. This *hepatic fever* is generally ushered in by a violent chill, and the paroxysms, which are repeated at regular times, are apt to be followed by increased jaundice. Their irregularity,—to which, however, there are exceptions in the earlier part of the case,—their resistance to quinine, the frequent occurrence of vomiting and of pain in the region of the liver, the history of the case, and the absence of malarial corpuscles in the blood, distinguish them from malarial fever. From abscess of the liver the affection is more difficult to discriminate, and we must lay stress on the deep jaundice, which mostly happens after the fever outbreaks, and on the different physical phenomena. Sweats occur in both, but they occur only at the end of the marked paroxysms in the so-called hepatic fever. We also find similar attacks of rigor and intermittent pyrexia associated with hepatic pain in obstruction of the common bile-duct from gall-stones. They may go on for years, and lead to death by anæmia and exhaustion, or recovery may take place. The temperature between the attacks is normal. Charcot

[1] Heitter, Wien. Med. Wochenschr., 1887; Margotta, Rivista Veneta, Feb. 1897; Favero, Gazz. degli Osp., Jan. 1899.

looks upon them as septic, as do Pepper[1] and Osler;[2] Ord[3] holds the fever-outbreak to be due to irritation of the mucous membrane, and this is the view I hold. Hepatic fever bears a close relation to malaria. Those who have malarial poison in their systems are more liable to it, and it is likely to be in them connected with a biliary catarrh, and with inspissated bile rather than with an impacted gall-stone.

Now, considering the question of operative interference that may arise, it is of the utmost importance to distinguish the cases in which the obstruction is purely catarrhal and not connected with gall-stones from those in which it is. Distention of the gall-bladder will not assist us, certainly not in recognizing the obstruction of the common duct from stone, for Ecklin has found that in nearly all cases the gall-bladder is contracted. The most certain test undoubtedly would be having found gall-stones on previous occasions, or finding them after the fever paroxysms. The cases with gall-stones are very much more frequent than the cases of hepatic fever without them; the jaundice is more distinctly connected with the attacks, and generally passes off more completely between them; the pain is greater and ceases more abruptly; and the febrile paroxysms are not brought on by cold, exposure, and fatigue, as they are often in hepatic fever without gall-stones.

Acute Cholecystitis.—This may be the result of the irritation of gall-stones, but often it is not, and is due to bacillary infection of the gall-bladder, particularly in typhoid fever; it is also observed as a sequel to pneumonia. At times we meet with an acute attack where chronic inflammation of the gall-bladder is present, but where every now and then there are acute outbreaks. Such cases may exist with gall-stones, but I have known them without these, especially in persons with chronic constipation. In some instances of acute cholecystitis the cause remains obscure.

The disease occurs in three forms, that are not, however, very sharply defined in their differences: the catarrhal, the suppurative, and the phlegmonous. The most significant symptom in all is the severe pain. This often occurs in violent paroxysms, and is referred to the seat of the gall-bladder, or is higher up, or epigastric, or near the appendix. It is nearly always associated with marked tenderness, which is not confined to the region of the gall-bladder, for the whole abdomen may become very sensitive. Yet the tenderness is apt to localize itself finally over the gall-bladder. There is often

[1] Medical News, March 29, 1890.

[2] Johns Hopkins Hospital Reports, vol. ii., No. 1, 1890.

[3] Boston Medical Journal, 1887.

coexisting rigidity of the abdominal walls, especially on the right side. The temperature is but slightly elevated. Jaundice is oftener absent than present. I found it in twenty of sixty-one cases I analyzed, and, when present, it is mostly slight, though before pronouncing it absent the urine must be carefully examined for bile pigments. Nausea and vomiting are more constant. Constipation is the rule, and the stoppage of the bowel may be so complete as to be looked upon as due to acute obstruction, and lead to an operation.

Tumor is as valuable a sign of acute cholecystitis as pain, though not so usual a one. It is at the seat of the gall-bladder, and is found at the junction of the upper two-thirds with the lower third of a line drawn from the ninth rib to the umbilicus,—at Mayo Robson's point.[1] The seat of the sensitive swelling is very significant in distinguishing acute cholecystitis from appendicitis. But where this affects the upper part of the appendix, there are tenderness and swelling high up, and not, as usual, in the right iliac fossa, over or near McBurney's point. and an absolute differential diagnosis may become impossible. In three cases reported in the admirable paper of Maurice Richardson[2] an operation was performed for appendicitis, which from the symptoms appeared positive, and a distended, inflamed gall-bladder was found. The swelling and the acute symptoms may suggest the possibility of abscess of the liver near the site of the gall-bladder, but there is much more pain in cholecystitis, and the chills, the irregular fever, the sweating of abscess are wanting.

There is no certainty in the diagnosis between the different forms of acute cholecystitis. The suppurative form is most commonly found in association with gall-stones, though we also meet with it in infective fevers, especially typhoid fever. The empyema of the bladder often attains considerable size, and a marked tumor is found descending with inspiration. The suppuration is not always limited to the gall-bladder. Marked leucocytosis points to the gall-bladder lesion being suppurative. Acute phlegmonous cholecystitis—the acute progressive empyema of Courvoisier—runs generally a very rapid course, and leads to death by general peritonitis, with which it is apt to be confounded. The action of the bowels is paralyzed, and intestinal obstruction is thus closely simulated.[3] Phlegmonous cholecystitis may lead to perforation of the gall-bladder with symptoms of collapse.

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[1] Diseases of the Gall-Bladder and Bile-Ducts, 1897.

[2] Acute Inflammation of the Gall-Bladder, Amer. Journ. Med. Sci., June, 1898.

[3] See case of Arbuthnot Lane, Lancet, Feb. 1893, in which there was an operation followed by recovery.

Acute Cholangitis.—Inflammation of the bile-ducts may be associated with any of the forms of inflammation of the gall-bladder, or exist without it. Most commonly it is of infective origin, due to gall-stones in the common duct, or to influenza, or to the bacillary infection of typhoid fever. There is general tenderness of the liver with or without pain, enlargement of the liver, and an irregular fever which takes the form of hepatic fever; and usually a persistent, slight icterus between the marked attacks. The disease may continue for a long time, and in the suppurative form is generally subacute in its course. There is in this great loss of strength, anæmia, and marked emaciation; and a septic endocarditis, or pleurisy or pneumonia may lead to death.

Acute Diseases characterized by a Decrease in the Size of the Liver and by Deep Jaundice.

Acute Yellow Atrophy.—This dangerous affection consists in a rapid diminution of the liver, with disintegration in the secreting-cells. To this disease belong most of those cases of malignant jaundice which terminate rapidly in death after violent cerebral symptoms. The malady scarcely ever lasts a week; generally a few days only elapse before the patient becomes comatose and dies.

The complaint is sometimes ushered in by nausea, a coated tongue, irregular action of the bowels, and a frequent pulse; at other times it begins abruptly with pain in the head, and with vomiting, at first of the contents of the stomach, but soon of coffee-ground material, which is evidently altered blood. The skin is yellow, and becomes from hour to hour more discolored. Jaundice is, indeed, never absent: it may not make its appearance before the other urgent symptoms, but sometimes it precedes the signs of serious difficulty for several days, or even for longer,—perhaps for upward of two weeks.[1] That the jaundice is not due to obstruction is proved by the stools containing bile. There are not uncommonly pain at the epigastrium and in the hepatic region, vomiting, muscular and arthritic pains, dyspnœa, meteorism, enlargement of the spleen, epistaxis, and hemorrhage from the bowels. The pulse exhibits extraordinary changes: it is generally very rapid, but sinks at times, without any assignable reason, to a normal frequency; during the deep coma of the last stages of the malady the beat of the artery is apt to become slow and full, but it may be quick and small. There is fever, not, however, active; the temperature may be, indeed, after the early stages of the disease,

[1] As in Observation No. XVII. of Frerichs on Diseases of the Liver.

below the norm until towards the end, when it has been known to be 104° or 105°. The surface may be covered with petechiæ. But, if we except the deep jaundice and the lessening hepatic dulness, the most significant symptoms are those referable to the nervous system. Severe headache, delirium, involuntary discharges, tremors, spasms, convulsions, or a constantly increasing stupor with sluggish pupils, show clearly what disturbance the poisoned blood is creating in the nervous centres.

Acute atrophy of the liver rarely happens in children or after forty years of age; it is much more common in women than in men. We find it not unusually following violent mental emotions or drunkenness and venereal excesses; or it occurs during pregnancy, and is then accompanied by renal disorder.

In point of diagnosis we have chiefly to consider the distinction from yellow fever, and from such diseases as typhoid fever, peritonitis, pneumonia, and meningitis, when accompanied by jaundice and delirium. The character of the eruption, the presence of diarrhœa instead of constipation, the milder nature of the mental wandering, the significant temperature record, the slight icterus, the Widal reaction, and the slower progress of the disease are of much value in enabling us to distinguish between *typhoid fever* and the typhoid symptoms of acute yellow atrophy of the liver. From *yellow fever*, acute atrophy differs by the epidemic character of the former, by the injected eye, by the intense pain in the back, limbs, and forehead, by the stages the febrile malady presents, by the decided fever temperature, by the usual presence of markedly albuminous urine, by the comparative absence of cerebral symptoms, and by the enlargement rather than the lessened size of the liver.

From the other affections named, the hepatic disorder may be discriminated by a thorough examination of the various organs of the body, and by a careful weighing of all the symptoms. In truth, it is thus only that we can avoid error; since, unless we can establish the most positive sign of acute atrophy, the diminution of the area of percussion dulness of the liver,—and there are cases in which we cannot establish this, particularly if there have been enlargement from previous disease,[1]—there is no manifestation of the hepatic malady that may not occur in the diseases mentioned, when they are complicated by jaundice. It is true that vomiting of blood is scarcely among their symptoms; but this does not invariably happen in acute atrophy. In cases of doubt we may be influenced by the presence of tyrosine and

[1] As in a case in my ward at the Pennsylvania Hospital.

leucine in the urine; and by the test for urea, which is greatly reduced or absent. So may be the uric acid, the chlorides, the sulphates, and the earthy phosphates. We may in this connection remark that leucine and tyrosine have been also found in the blood and in many tissues. This was observed in a case which I saw with Dr. H. C. Wood, and which he has carefully reported.[1] On the other hand, one or both may be wanting in undoubted cases of acute atrophy. Tube-casts and small quantities of albumin are not uncommon.

An affection like acute yellow atrophy occurs from phosphorus poisoning; and indeed there are those who believe that acute yellow atrophy is really due to phosphorus accidentally introduced into the system.[2] The occurrence of the fatal malady in pregnant women has already been referred to. Jaundice from mental emotion, or produced by the pressure of the gravid womb, is in them not unusual; and we may be called upon to distinguish this harmless form of icterus from that of yellow atrophy. In the serious derangement of the nervous system, and the graver character of all the symptoms, lie the marks of separation.

Chronic Diseases attended with Enlargement of the Liver, and with slight or no Jaundice.

Chronic Congestion.—This morbid condition is observed chiefly in persons of sedentary habits, or in those who indulge too freely in the pleasures of the table, or use large quantities of alcoholic drinks or fermented liquors. It is frequently met with in hot climates and in malarial districts. It may also occur in scurvy, and in connection with abdominal affections which interfere with the portal circulation, or it may happen in consequence of a disturbance of the flow of blood through the liver, dependent upon disease of the heart.

Whatever the source of the hyperæmia, the symptoms are similar. They are impaired appetite, bitter taste in the mouth, a coated tongue, flatulency, a feeling of tension and weight in the right hypochondrium, depression of spirits, loss of strength, impoverishment of blood, deposits of lithates from the highly colored urine, headache, dry cough, and occasional nausea and diarrhœa, or looseness of the bowels alternating with constipation, and, in protracted cases, hemorrhoids. The conjunctiva has constantly a more or less jaundiced

[1] Amer. Journ. Med. Sci., April, 1867.

[2] Perls, Handb. d. Allg. Pathol., i., points out an anatomical distinction; in acute atrophy there is fatty degeneration; in phosphorus poisoning the liver-cells are only infiltrated with fat.

tinge; the dulness on percussion in the hepatic region is increased in extent; at times the enlarged liver pulsates. In some cases the habitual congestion leads to an altered condition of the bile-ducts and of the secreting-cells of the liver; but ordinarily, unless the hyperæmia be kept up by some exciting cause which it is impossible to remedy,—such as an abdominal tumor, or an organic affection of the heart,—it can be removed. A troublesome feature of the malady is its disposition to return.

Chronic hepatic congestion is sometimes confounded with, or rather there is mistaken for it, a liver which has been pushed downward by the habit of *tight lacing*. But the absence of any signs of hepatic derangement, and the lowered outline of the upper border of the displaced right lobe, enable us to distinguish this state.

Chronic hepatic congestion, as indeed any disease of the liver which leads to its enlargement, may be confounded with *chronic gastric catarrh*. But the outline of the dulness when the liver is increased in size, the jaundiced hue of the conjunctiva, the altered character of the stools, and the less marked gastric symptoms will enable us to arrive at a correct diagnosis. Yet we must not forget that the two morbid states are often conjoined.

Hypertrophy of the liver may present the manifestations of congestion. The little we know of an increased formation of the liver-cells teaches us that this may happen as a partial hypertrophy, to compensate for loss of substance, in instances in which a portion of the gland has been destroyed; or, as a more general increased growth, in diabetes, in leukæmia, and as a consequence of malaria. Yet there is never any certainty in the diagnosis.

So-called *torpor of the liver*, in which there is supposed to be a deficient excretion of bile, has much the same symptoms as congestion. In persons of middle life who eat freely and take too little exercise in the open air, or those of sedentary habits in whom anxiety and worry have lowered the nervous tone, the well-known symptoms of headache, languor, depression of spirits, loss of appetite, drowsiness after meals, sallow hue of skin, dingy conjunctiva, urine depositing lithates, stools black and offensive, or more often pale or whitish, bespeak this "bilious" state, and we can only distinguish the functional disorder from the ordinary forms of chronic congestion by the history, the concurrent symptoms, and the enlargement of the organ, which these present.

The symptoms of chronic congestion of the liver, as of other hepatic derangements, show themselves at times more particularly in the *nervous system*. Headache, vertigo, dimness of sight, and noises

in the ears are common; and tingling and prickling sensations and a feeling of creeping in the extremities may cause needless fear that paralysis is imminent, and disappear under a mercurial and a few saline purgatives. On the other hand, signs of stomach and liver derangement may be really due to an affection of the nervous system. Twice it has come under my observation that altered character of the stools, bitter taste in the mouth, vomiting, and slight discoloration of the conjunctiva, existing in connection with tumors at the base of the brain, were considered as purely of hepatic origin. Clifford Allbutt[1] cites a case of Ménière's disease, in a physician, where the vomiting and giddiness were thus wrongly accounted for. In such instances, the disordered gait, persistent noises in one or both ears, and the loss of power of hearing of one ear, shown when a tuning-fork is placed in contact with the skull on the affected side, tell the true meaning of the other symptoms.

Chronic Hepatitis.—It is difficult to say what are the symptoms of the malady, because most of the chronic affections of the organ, especially the congested, the fatty, the albuminoid liver, and hypertrophic cirrhosis have been included in its description. The liver is enlarged in size. The inflammation may be chronic almost from its onset, and be developed under much the same circumstances as chronic congestion; or it may succeed to acute hepatitis. But chronic hepatitis is not a common disease, and is scarcely to be distinguished from persistent hyperæmia of the organ, unless, as so often in tropical hepatitis, abscess result.

Abscess of the Liver.—In temperate climates we seldom encounter this affection, save as the consequence of an embolic or pyæmic process in the liver, or in connection with some disease of the intestines, or of abscesses around the rectum, or as a sequel of gastric ulcer, or of pylephlebitis, or of gall-stones which have produced ulceration of the gall-bladder and gall-ducts and secondary abscesses of the liver, or of traumatism, or of suppurative disease of bones. In hot climates it is not an unusual disease, both in connection with dysentery and without it.

The symptoms of hepatic abscess are obscure. Sometimes the only symptoms are debility, great irritability of the nervous system, and irregular slight febrile attacks. More usually the formation of pus gives rise to rigors, leads to night-sweats, and not infrequently to the development of a fever simulating that of a quotidian or tertian intermittent or remittent, and attended during certain hours of the

[1] St. George's Hosp. Rep., vol. viii.

day with considerable elevation of temperature. Jaundice occurs, but is generally slight, and is often absent. There is no enlargement of the abdominal veins, nor is there, save exceptionally, ascites or œdema of the lower extremities. Dry cough, quickened breathing, and gastric disorder, especially loss of appetite, are frequent, and obstinate vomiting, hiccough, and meteorism are not unusual. There is always marked leucocytosis. In the advanced stages of the malady typhoid symptoms are apt to develop. But the disease may be latent. The local signs, too, are far from being always obvious, or indeed uniform. In some instances the hepatic region is more prominent than natural, and we can detect fluctuation over portions of the enlarged gland; but neither sign is constant, and the latter depends greatly upon whether or not the abscess is deeply seated. Tenderness, either general or limited, is found only in a certain proportion of cases, especially when the abscess is near the surface. It is frequently associated with a throbbing or a dull pain, which may be transmitted to the right shoulder. According to Annesley,[1] this sympathetic pain in the right shoulder indicates that the convex part of the right lobe of the viscus is affected. Conjoined to the feeling of weight, and to the throbbing in the hepatic region, is at times a tension occasioned by palpation of the abdominal muscles, especially of the rectus. Twining[2] regards this as very significant of deep-seated abscess. The pain of hepatic abscess may be acute, like that of an intercostal neuralgia, and greatly aggravated by cough.[3] Cyr[4] tells us, with reference to the exact position of the abscess, that when it is in the front convex part of the liver there is pain radiating to the chest and shoulder, dyspnœa, but rarely jaundice; when in the central part of the organ, there are few signs of local affection of the liver itself or adjacent organs, except decided jaundice if the abscess be large. In abscess limited to the under surface, thoracic symptoms are absent, but gastric symptoms, especially uncontrollable vomiting, occur; the pain is apt to radiate towards the groin.

A positive diagnosis of abscess of the liver is often a very difficult matter; for there are a number of affections with which it may be readily confounded. Prominent among these are hydatids, cancer of the liver, actinomycosis of the liver, affections of the gall-bladder, and a pleuritic effusion on the right side.

From *hydatids* of the liver, the febrile symptoms, the disturbed

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[1] Researches into the Diseases of India. [2] Diseases of Bengal.

[3] Malbot, Abcés du foie en Algerie, Arch. Gén. de Méd., Aug. 1899.

[4] Traité des Maladies du Foie, 1887.

nutrition, and the pain distinguish an hepatic abscess, except in those cases in which the cyst becomes the seat of suppuration. Under these circumstances error can scarcely be avoided, unless we are fully cognizant of the previous history.

Cancer of the liver differs from an abscess by its dissimilar history, by the hard nodular masses, and by the absence of fluctuation. It is only in rapidly growing medullary cancer that we can discern a sense of fluctuation; but even here we can generally distinguish some nodules which do not fluctuate. Further, the marked fever and the other constitutional symptoms are not like what occur in hepatic cancer; for in this affection, as in all cancers, the temperature, except in instances of large, rapidly spreading growths, is but little affected,—may, indeed, be subnormal.

Actinomycosis of the liver may give rise to a collection of pus, and the abscess may discharge through the loins or through the lungs, as in hepatic abscess. The hepatic swelling is painful on pressure, but is unlike that of hepatic abscess in arising suddenly from the parts beneath, and in being surrounded by a firm base in the liver. These characters distinguish it from an ordinary abscess as well as from hydatid of the liver.[1] Yet it is by the history, and by finding the ray fungus in pus from other diseased parts of the body, that the diagnosis is mostly established, for actinomycosis of the liver is almost never primary.

Of the *affections of the gall-bladder*, the one most liable to be confounded with hepatic abscess is distention. This occurs either from a closure of the cystic or of the common duct, especially the former, or from cholecystitis, with perhaps a subsequent closure of the ducts. In such a case the gall-bladder may become enormously distended with decomposing bile and puriform matter, and thus may be occasioned a fluctuating tumor, tender on pressure, and readily mistaken for an abscess. Now, we are sometimes able to distinguish the soft swelling caused by a diseased gall-bladder by its situation, its pear-shaped form, its mobility, its distinct and persistent fluctuation; by the normal appearance of the parietes of the abdomen; by the tenderness over the tumor and absence of tenderness over the liver; and by the fact that affections of the gall-bladder are frequently preceded by repeated attacks of violent pain due to the passage of biliary calculi. Then we find little jaundice, or none at all; and no hectic fever. But to neither of these circumstances can we trust implicitly. For there is apt to be intense jaundice in an affection of the gall-

[1] Harley, Med. Chir. Transact., vol. lxix., 1886.

bladder, if the common duct also be implicated; and jaundice is, in abscess of the liver, a symptom more frequently absent than present. And with reference to hectic fever, the continued suppuration in the distending sac may produce it, and lead, indeed, to great constitutional disturbance.[1] Further, these *biliary abscesses* may, like hepatic abscesses, open externally, or burst into the chest. At times the communication is with the bronchial tubes, and gives rise to very anomalous symptoms. Thus, Simmons[2] details a case in which there was a tumor in the epigastrium, fluctuating, with a sense of intervening air or gas, and resonant on percussion; a blowing sound was distinctly discerned synchronous with the respiratory act, and occasionally accompanied by a gurgling noise; there were no signs of pneumothorax. At the autopsy a biliary abscess was found communicating with the right bronchus.

A *pleuritic* effusion on the right side is distinguished from an hepatic abscess by the physical signs of the effusion. But abscesses of the liver may open into the right pleural cavity. Then we observe the physical signs of a pleuritic effusion subsequent to those of hepatic abscess. Finally, it generally happens that large quantities of purulent sputa are expectorated; in rarer instances the pus is discharged through the walls of the chest. In the former case, the accumulation of pus in the pleura may be limited; the inflammation of the pleural membrane may be circumscribed, while the signs of an inflammation at the lower portion of the right lung, dulness on percussion, tubular breathing, and rusty-colored sputa, are evident. These phenomena may subside, and the respiration in parts become inaudible, when a discharge of a large quantity of a reddish or whitish pus takes place, in which the elements of bile and the microscopical appearances of the hepatic tissue may be detected. Gradually this expectoration ceases, and the affected textures heal. But in some instances the discharge never stops, and the patient dies worn out by the constant drain.

In *subphrenic peritonitis* the exudate may occasion a swelling and lead to an abscess producing misleading symptoms. The tumor shows itself chiefly in the left hypochondrium or the epigastrium, and seems to disappear when the stomach is distended with gas, and to increase when the stomach is full; the colon always lies below the tumor. The constitutional symptoms are those of suppuration; the chills and irregular fever may be very marked symptoms; there is

[1] As in a case reported by Pepper, the elder, Amer. Journ. Med. Sci., Jan. 1857.

[2] Amer. Journ. Med. Sci., Oct. 1877.

much pain, vomiting, and embarrassed breathing. The *subphrenic abscesses* develop generally as the result of perforation of a gastric or duodenal ulcer. They are very apt to be mistaken for abscess of the liver, and, except by their history and the characters mentioned, cannot be discriminated. These too, chiefly distinguish them, when they also press upward, from a collection of fluid in the right pleural sac. They often contain air, extend into the thorax, and we then have developed that curious condition described as *subphrenic pyopneumothorax*, which, when on the right side, is, except for the physical signs, easily mistaken for the breaking of an hepatic abscess into the chest. The history of the affection is generally significant; the subphrenic abscess itself is the result of a perforating ulcer of the stomach or of the duodenum, occasionally of an appendicitis, and at times is preceded by the symptoms of a general or local peritonitis or by the discharge of pus from the bowels, and it sets in abruptly with pain and vomiting of bilious or bloody kind. The tumor formed by the subphrenic abscess has the characters just described. The signs of pneumothorax subsequently show themselves, as Leyden[1] has found, with distinct metallic tinkling and succussion sound. Yet, while all breath-sound is sharply cut off below the fourth or fifth rib, up to this point the normal vesicular murmur is heard on deep respiration, and there are no signs of pressure in the pleural cavity or of distention of the chest; and the marked alteration, by change of position, of the dulness on percussion, from the exudation at the lower part of the chest, is strictly limited to this part. The liver reaches to the umbilicus or lower, and when a canula is passed into the cavity beneath the diaphragm and a manometer is attached, inspiration shows increased pressure, expiration the reverse,—exactly opposite, therefore, to what happens if the canula be in the pleura.

When an hepatic abscess forces its way *externally*, it may, prior to its discharge through the thoracic or abdominal walls, occasion difficulty in diagnosis from abscesses originating in these walls. Nothing but a careful consideration of the attending symptoms and of the history of the case will lead to a differential distinction. Nor does the difficulty wholly cease when the slowly developed tumor, which an hepatic abscess forms, has opened: since it is far from always that we find in the pus the evidences of the broken-down liver-tissue, and it is only occasionally that the fluid is of yellow or greenish color and yields the reactions of bile. The means of discrimination most to be relied upon is a probe: for by the depth to

[1] Zeitschrift für klin. Med., Bd. i.

which it can be passed, the direction it takes, and the feel of the structures it encounters, we are placed in possession of many important facts. In doubtful cases, also, we employ the aspirator, and a chemical and microscopical examination of the pus, other than that oozing out of the opening, may tell the nature of the abscess. Indeed, the aspirator may be made a means of diagnosis of abscess of the liver under some of the circumstances above mentioned, where abscess is closely simulated by other hepatic affections. No harm results from the exploration, even if no abscess be found.

Occasionally a *hernia through one of the recti muscles* is mistaken for a projecting abscess of the liver. I was called some years since to see such a case, in which the opinion that it was an abscess of the liver had been long entertained. The sound of the mass on percussion; the clearly defined limits of the liver; the absence of hepatic and gastric symptoms,—taught the true nature of the malady.

Much has been said of the distinction between the abscesses which are developed in the course of embolism or of pyæmia, "the pyæmic abscess," and the abscess, common in tropical climates, which forms as the result of hepatitis, "the tropical abscess." This kind of abscess is often met with following dysentery. One of its forms occurs in connection with the amœba coli, though we may have abscess of the liver due to the amœba without dysenteric symptoms, and tropical abscess irrespective of any kind of dysentery. There is first a pathological change in the liver, and then, it is supposed, a microbic infection.[1] The points of distinction between pyæmic and tropical abscess may be thus tabulated:

Pyæmic Abscess.	Tropical Abscess.
Many in number; small in size.	Usually a single large abscess, seated in right lobe, towards the convexity of the liver.
Uniform enlargement of liver; only exceptionally bulging of ribs.	Enlargement not uniform; bulging of ribs, or in epigastrium, or in right hypochondrium.
No fluctuation; always pain and tenderness.	Fluctuation usual; pain and tenderness always absent.
Jaundice present in the majority of cases.	Jaundice exceptional.
Enlargement of spleen usual.	Enlargement of spleen unusual.
Rigors and night-sweats marked; often symptoms of blood-poisoning.	Rigors and night-sweats less marked; obstinate vomiting often present.

[1] Davidson, article "Suppurative Hepatitis," Allbutt's System of Medicine, vol. iv.

Pyæmic Abscess.	Tropical Abscess.
Course rapid; three weeks to three months.	Course less rapid: often extends to three or six months, or longer.
Arises after external injuries and operations, or suppurating cavities, or ulcerations, such as ulcers of the stomach or gall-bladder.	Arises in tropical climates, chiefly in those who eat and drink largely: dysentery frequently coexists.

Fatty Liver.—A fatty liver occurs in drunkards: in obese persons; in wasting diseases, especially in phthisis; in the course of protracted diarrhœa, and sometimes in children after exanthematous fevers.

A knowledge of the sources of fatty liver is the most important element in the diagnosis; for neither the physical signs nor the symptoms present anything which is characteristic. The physical signs are simply those of an enlarged painless liver; the enlargement is generally moderate and uniform, and the lower margin rounded. The symptoms are much the same as those of hepatic congestion, except that there is perhaps greater tendency to diarrhœa. There is no ascites; the amount of jaundice is always very slight; in truth, jaundice is most frequently wanting.

Waxy Liver.—This peculiar degeneration of the liver which forms part of a general cachexia manifests itself rather by the signs of disturbance of other organs than by the direct proof of altered function of the viscus affected. Thus, disordered digestion, nausea, vomiting, tympanites, discolored stools, and diarrhœa are much more frequent than jaundice, which, indeed, is very much oftener absent than present. There is a feeling of fulness in the hepatic region, but no pain: while physical exploration exhibits an increased percussion dulness, and shows the dense organ to have a well-defined though somewhat rounded margin. The enlargement is uniform, but considerable: at times so great that the liver occupies a large part of the abdomen, producing a visible bulging. The smoothness and the regularity of outline are lost if waxy liver coexist with diseases of the liver which may harden the organ in nodules, such as cancer, fibroid changes, or cirrhosis.

Enlargement of the spleen is commonly associated with the enlargement of the liver, and in many cases the urine is albuminous from waxy disease of the kidneys. Dropsy, as a rule, is not encountered: but in this respect much depends upon the state of the kidneys and of the blood, or upon the existence of secondary peritonitis.

Waxy liver is much more common in males than in females. It is usually caused by constitutional syphilis or coexists with scrofulous diseases of the bones, with unhealed ulcers, especially rectal ulcers, with long-continued suppuration. In some instances it is associated with cancer or with phthisis, or malaria, or results seemingly from the abuse of mercury. There is always a cachexia.

The disease is one lasting for years. In advanced cases, besides the spleen and the kidneys, the stomach and the intestines are apt to be implicated; looseness of the bowels, with dysenteric symptoms arises, and the skin and breath have a musty, disagreeable odor.

Now, when we contrast a waxy liver with other hepatic complaints in which the liver is enlarged, we find it resembling most closely the *fatty* and the *syphilitic* affections. But in the former, although there is enlargement, it is not often so great as in the waxy liver. Besides, the organ feels softer on palpation, and the disorder is not associated with a diseased spleen or kidney, and is much less likely than a waxy liver to give rise to dropsy. Then the history of the case is very significant. A syphilitic hepatitis, with which indeed the waxy liver is at times combined, is further distinguished by the prominent nodules felt on the surface of the liver. From *congestion of the liver*, waxy liver is readily discriminated. A comparatively slight affection in which jaundice is frequent is very different from a malady in which the hepatic disease is but part of a general morbid state and in which jaundice is very infrequent. In *leukæmic liver* we may have considerable and smooth enlargement, but the history of the case and an examination of the blood tell its true nature.

Cancer of the Liver.—In cancer of the liver the organ is almost invariably large, and sometimes it reaches an enormous volume. It is irregular and uneven, nodules of various size being developed in its substance and projecting from its border and surfaces. These prominences are harder than the surrounding hepatic tissue; but there are exceptions to this rule, for sometimes, especially in the encephaloid variety, the elastic tumors impart, when pressed, a very deceptive sense of fluctuation. The cancerous masses increase, and in some cases with great rapidity.

The malignant disease is rarely confined to the liver; it frequently supervenes upon cancer of the mammary gland, or of the uterus, or of the stomach, or pancreas. It is an affection of middle life or of old age; yet it occasionally occurs in young persons. I have met with two cases of primary cancer of the liver in women not twenty-five years of age, and two in children. In primary cancer of the liver we generally find a history of cancer in the family; and pro-

tracted grief or anxiety, Murchison tells us,[1] may precede the development of the malady, whether a family taint can be traced or not. Cancer of the liver rarely lasts beyond a year, and it may run a rapid course. This is especially the case with primary cancer. The proportion of this to secondary cancer is stated by Hale White[2] as one to twenty-five.

In the diagnosis of hepatic cancer, the most important physical signs are the increased percussion dulness in the hepatic region and the uneven surface detected on palpation. The enlarged liver is found extending across the epigastrium far into the left hypochondrium; it reaches at times lower than the umbilicus, and presses the diaphragm upward; the line of dulness moves markedly downward with full inspiration. The nodules can often be felt distinctly through the abdominal walls, and deep inspiration may reveal a nodule otherwise not perceptible. The diseased organ is painful, and tender to the touch. In cases in which the peritoneal covering is affected, the tenderness is greatest. And, although any of these three phenomena—the enlargement, the uneven surface, and the tenderness—may be absent, they are tolerably constant attendants on cancer of the liver. The tenderness is rarely wanting.

Among the symptoms of hepatic cancer, we find gastric and intestinal disturbances; pain in the right shoulder; an annoying cough; rigidity of the abdominal muscles; wasting of the whole body; a cachectic look; occasional febrile attacks, yet, on the whole, normal or subnormal temperature; and, in the later stages, sometimes hemorrhages from the stomach or bowels, and diarrhœa. Ascites, too, is observed, and is generally dependent either upon chronic peritonitis attending the development of the cancer, or upon the pressure this exerts upon the larger branches of the portal vein. Jaundice may or may not be present; it is frequently wanting. I have seen it intense when the cancerous growth or a cancerous gland pressed on the bile-ducts, and sometimes it is of a peculiar dark-green color. In any instance it persists until death. There are cases in which all these symptoms are perceived; in others only some occur, and in others, again, even these few may not be well defined. Indeed, when we consider the amount of deposit which is generally present; when we regard its character; when we take into account the necessarily impaired function of one of the most important glands in the body; when we reflect upon the pressure which the enlarged organ must

[1] Lectures on Diseases of the Liver, 2d edit.

[2] Tumors of the Liver, Allbutt's System of Medicine.

occasion,—it is truly astonishing that often so little dropsy, so little jaundice, so little pain, so little constitutional disturbance, are produced by the disease.

Yet in point of diagnosis we can generally discern the malady by the combination of the symptoms and signs indicated. It is only at an early stage of the disease, or when the liver is not enlarged, that we are apt to be in doubt. When the liver is the seat of cancer, but is not increased in size, the recognition of the malady is next to impossible. In these obscure cases, the persistent tenderness in the hepatic region, accompanying the evidences of disturbed function of the liver, ascites, anæmia, and a cachectic appearance, are the signs most likely to lead to a correct conclusion. In any instance, jaundice coming on in a person over forty years of age, lasting for months, and associated with gastric disease and failing health, must, in the absence of a history of gout or of syphilis, be looked upon as pointing to hepatic cancer, if we can exclude cancer of the pancreas. Again, we must remember that loss of flesh and of strength often precedes jaundice and pain,—in fact, all signs of disorder of the affected organ.

Let us pass in review the complaints with which well-marked cancer of the liver may be confounded. Omitting, because elsewhere discussed, hydatids, abscess of the liver, and hypertrophic cirrhosis, they are:

WAXY LIVER; FATTY LIVER; CHRONIC CONGESTION;
ACUTE CONGESTION; ACUTE HEPATITIS; CATARRHAL JAUNDICE;
SYPHILITIC LIVER;
AFFECTIONS OF THE GALL-BLADDER;
CANCER OF THE STOMACH;
CANCER OF THE OMENTUM;
ENLARGEMENT OF THE RIGHT KIDNEY.

Waxy Liver; Fatty Liver; Chronic Congestion.—A waxy liver presents often as much increase in size as cancer; moreover, like cancer, it is associated with evident signs of cachexia. The main points of distinction are the smooth surface and uniform increase of the liver in waxy disease, its painlessness and slow progress, its combination with enlargement of the spleen and markedly albuminous urine, and the history of the case pointing to long-continued suppuration, to constitutional syphilis, or to diseases of the bones, or, in fact, to one of the causes which generally lie at the root of waxy degeneration. In the differentiation of cases of infiltrated cancer without distinct nodules, the physical exploration does not aid us, and we have to lay stress on the other points.

A fatty liver is easier to discriminate from hepatic cancer. The occurrence of the non-malignant malady in the obese, in consumptives or in drunkards, and the total absence of pain,—in truth, of any decided indications of hepatic disease, except increased size of the organ,—enable us to distinguish between the two affections. The slighter signs of disturbance, both constitutional and local, the dissimilar history, and the uniform enlargement of the liver separate chronic congestion from cancer. As a mark of distinction, too, of the cancerous from all of these non-malignant disorders, Virchow lays stress on the existence of swollen jugular glands; and a small cancerous induration in the abdominal walls, around the umbilicus, also not infrequently aids the diagnosis.

Acute Congestion; Acute Hepatitis; Catarrhal Jaundice.—It is rarely indeed that these ailments are confounded with cancer of the liver, because the history and the course the latter malady takes are so dissimilar to those of an acute hepatic disorder. Yet there are cases in which the malignant disease is either developed with great rapidity, thus simulating an ordinary acute affection, or has lain dormant and passed unnoticed until it begins suddenly to increase. Under such circumstances we may be able to recognize the malignant complaint, if its physical phenomena be well defined; but if these be not clearly marked, the diagnosis is one of great difficulty.

To cite a case in illustration: A married woman, twenty-five years of age, was admitted into the Philadelphia Hospital on January 14, 1862, with jaundice and slight fever. She stated that she had been in excellent health until about two weeks before, when she caught cold by sleeping in a damp apartment. Her appetite and digestion had been good previous to her present illness, and she had been fully able to perform her household work. Since she was taken ill she had noticed a feeling of weight in the region of the stomach and liver. Râles indicative of bronchitis were found in the chest, and the impulse of the heart was feeble. The hepatic percussion dulness was somewhat increased in extent, especially that of the left lobe; but the outline of the organ appeared regular and even. Tenderness of the abdomen, more particularly in the epigastrium and right hypochondrium, was also noted. There was nausea, but no vomiting; the tongue was clean; the evacuations were discolored. Now, here was certainly a patient presenting none of the signs of hepatic cancer, except, perhaps, the tenderness over the enlarged gland. Yet at the autopsy, which was made within a week after her reception into the hospital, and therefore not three weeks from the apparent beginning of the complaint, whitish nodular cancerous spots, many of them soft,

were found in the substance of the liver, but not at its edges, nor forming anywhere distinct protuberances.

The similarity of certain cases of protracted catarrhal jaundice in elderly persons, presenting emaciation, with nausea, retching, and vomiting, has been above mentioned. The physical signs of the enlargement of the liver may or may not assist us, according to their character, but uniform enlargement without nodules and absence of marked tenderness would be in favor of the non-malignant view. The same points help us where inflammatory thickening about the biliary passages has happened in consequence of gall-stones.

Syphilitic Liver.—As a consequence of constitutional syphilis, the liver may at times exhibit cicatrices on its surface, and scattered nodules, consisting of connective tissue, and extending into the parenchyma. This condition is styled syphilitic inflammation of the liver, or the syphilitic liver. The organ becomes uneven from the contraction of the cicatrized parts, and is apt to be somewhat increased in size, from coexisting amyloid degeneration or interstitial hepatitis. The patient has a pale, cachectic look, but is not jaundiced,[1] except from a temporary catarrh of the bile-ducts; nor is dropsy present, unless there be at the same time an affection of the kidneys or enlargement of the spleen. But the most important elements in the diagnosis are the age of the patient, the history of the case, and the detection of syphilitic cicatrices in the throat. When contrasted with cancer, we find, besides these points, the chief distinctive marks to be: the much more usual absence of jaundice, of dropsy, and of pain, the increase in size of the spleen, the want of local tenderness,—unless this be due to passing attacks of perihepatitis,—the slow growth of the liver, and the smaller size and softer feel of the nodules. There are cases of syphilis of the liver in which an interstitial hepatitis is chiefly present, and which are scarcely to be distinguished from cirrhosis, except by the history and general evidences of syphilis. Syphilis of the liver may be hereditary.

Affections of the Gall-Bladder.—Dilatation and cancer of the gall-bladder are both very liable to be mistaken for cancer of the liver. The former affection may result from occlusion of the hepatic and common bile-ducts, or it may be owing to the distention of the bladder with an albuminous fluid,—the so-called dropsy of the gall-

[1] No jaundice is mentioned in the cases of Dittrich, Prag. Vierteljahrschr., Bd. vi. and vii.; of Gubler, Mémoires de la Société de Biologie, tome iv.; of Bamberger, Krankheiten der Leber, in Virchow, Pathologie, etc.; or of Moxon, in Guy's Hospital Reports, 1867. In the cases of Murchison, Diseases of the Liver, 2d edit., 1877, it was a passing or an absent symptom.

bladder. In either instance the bladder may attain an enormous volume, and give rise to a marked tumor at the lower margin of the liver. The prominence is apt to be rounded or pear-shaped, and, except in those instances in which the occlusion is in the cystic duct or at the neck of the gall-bladder, the impediment to the flow of bile is accompanied by intense jaundice and by decided hepatic swelling. In the uniform enlargement of the liver, the peculiar contour of the prominence, the absence of ascites, the paroxysms of pain preceding, not following, as in cancer of the liver, the other marked symptoms, and the history of the case, which not infrequently points to repeated attacks of colic from the passage of gall-stones, we find the clue which permits us to determine that we are not dealing with hepatic cancer. In reaching a conclusion we must, however, bear in mind that distention of the gall-bladder from secondarily enlarged cancerous glands pressing on the common duct often occurs.

Cancer of the gall-bladder is scarcely ever met with in young persons, and is, as a rule, associated with cancerous formations in the liver or in other organs. It is difficult to make out a certain diagnosis of the affection, for it presents a strong likeness both to cancer of the pyloric extremity of the stomach and to cancer of the liver. From the latter it is undistinguishable, unless the situation and form of the tumor be such that we can clearly recognize it as belonging to the gall-bladder. Sometimes it is preceded by a history of gall-stones.[1] Jaundice, as in cancer of the liver, may be absent or present: in five cases reported by Bamberger[2] it was found in all, and was even intense. Frerichs, on the other hand, states that in most instances it is wanting. Musser[3] finds it reported in sixty-nine out of a hundred cases. In sixty-eight out of one hundred cases analyzed by him a tumor was discovered, the position of which is most frequently in the right hypochondrium and the umbilical region, and which is painful on pressure. There is also gradually increasing pain and a sense of weight in the right hypochondrium. The disease is more common in women than in men. The signs of the cancerous cachexia are strongly marked; as a rule, more strongly than in hepatic cancer. In tumors affecting primarily the ducts, there is early and intense jaundice.[4]

Gall-stones occasionally accumulate in the gall-bladder in such numbers as to give rise to a hard, even nodulated swelling, which

[1] Murchison, *op. cit.*

[2] Krankheiten des Digestions-Apparates.

[3] Transact. Assoc. Amer. Phys., vol. iv., 1889.

[4] Rolleston, Med. Chronicle, Jan. 1896; Kelynaek, *ibid.*, Nov. 1897.

may be mistaken for cancer. But the tumor is generally movable, is not painful on pressure, and does not alter in size, or does so but slowly. Sometimes the patient complains of the feeling of a weight rolling from side to side when he turns in bed, and on palpation a crackling sound is produced, which is readily discerned with the stethoscope. Generally we obtain a history of bilious colic. There may or may not be jaundice; there is an absence of the cachectic symptoms of cancer. But we must always remember that gall-stones are frequently combined with cancer of the liver or gall-bladder.

Cancer of the Stomach.—This is discriminated from cancer of the liver by the far more constant vomiting, by the more obvious symptoms of indigestion, and by the persistent pain in the stomach. Moreover, the seat of the tumor is different; it is epigastric, or extending downward, but not often passing into the right hypochondrium, and it shows on percussion a very different contour from an enlarged liver. Yet there are cases in which we are kept in doubt; especially those in which the left lobe of the liver chiefly is affected with cancer and presses upon the stomach, inducing perhaps—and thus making the likeness still closer—obstinate vomiting. The only traits of distinction are then found in the presence or absence of marked derangement of the functions of the liver, and in the chemical examination of a trial meal.

Cancer of the Omentum.—The absence of jaundice, and the unaltered appearance of the stools, are here, too, of great value in indicating that a tumor near or joining the left lobe of the liver is not due to cancer of that viscus. Moreover, the boundaries of the morbid mass are different from those of a diseased liver. But we cannot always trust to this. Cancerous tumors of the lesser omentum may so surround the liver, and correspond so closely to the regular form produced by hepatic cancer, that the two maladies cannot be distinguished; at least not by the local signs. Again, a loop of intestine may be thrust across the enlarged liver at a point corresponding to the usual limit of the percussion dulness of its left lobe, thus dividing the most prominent nodules from the greater portion of the viscus, and making it appear as if the tumor were to the left of, and below, the stomach, and belonged, therefore, probably to the omentum.[1] In such cases we have to depend entirely upon the signs of disturbed liver function.

Enlargement of the Right Kidney.—A tumor formed by an enlargement of the kidney does not present the same outline of percussion

[1] See case, Proceedings Pathological Society of Phila., vol. i. p. 275.

dulness as a cancerous liver. The dulness is, moreover, surrounded by the tympanitic sound of the intestine, and is not lowered by a deep inspiration; and the signs of disturbed function of the kidney, and an examination of the urine, will generally materially assist the diagnosis. Still, cases may occasionally happen in which, owing to a peculiar shape of the diseased kidney and to the obscurity of the symptoms, an error in diagnosis can scarcely be avoided.[1]

Finally, in reviewing the diagnosis of cancer of the liver, we must inquire whether other than cancerous growths, such as sarcoma, melano-sarcoma, myxoma, epithelioma, cysto-sarcoma, angioma, lymphadenoma, can be distinguished from true cancer. They may produce identical physical signs and symptoms; indeed, a distinction is impossible, unless the history of the case and finding tumors elsewhere enable us to make it. Much the same may be said of that rare disease, tubercular formations in the liver. Leukæmic livers may attain enormous size, and be mistaken for cancer; and the cachexia that attends them makes the error more likely. But the swelling of the spleen and of the lymphatic glands and the microscopical examination of the blood furnish the points in diagnosis.

Hydatids of the Liver.—The development of one or of several cysts in the liver, containing within them echinococci, is not, as a rule, a disorder which occasions serious disturbance of the general health. Nor do the hydatids usually give rise to either jaundice, dropsy, or any marked signs of gastric or of intestinal irritation, or to fever, or to local pain. Their most constant manifestations are a decided increase of the size of the liver, and the presence of elastic tumors discernible in the hepatic region. In some instances xanthelasma has been noticed. This disorder of the skin, however, is not peculiar to hydatids, but has been observed in connection with other forms of hepatic enlargement associated with chronic jaundice. There is excretion of large quantities of urea.[2]

The growth of the hydatid is generally very slow, and usually in one direction only,—upward, downward, laterally. Very commonly the hydatid tumor grows from the right lobe. In most cases it attains considerable dimensions, and the liver may be found to encroach upon the lung as far as the second intercostal space, or to extend far down into the abdominal cavity. On percussion, the line of dul-

[1] Vidal (Bulletin de la Société Médicale des Hôpitaux, 1874) cites errors in diagnosis between tumors of the kidneys, especially hydronephrosis, and diseases of the liver attended with enlargement, like abscess or cancer, made by such masters in our art as Velpeau, Nélaton, Gosselin.

[2] Posselt, Deutsches Archiv für klinische Medicin, Bd. lxiii., 1899.

ness either of the upper or of the lower boundary of the viscus, or of both, is perceived to be very irregular, and occasionally on striking a series of abrupt blows we discern a peculiar vibration, similar to the sensation perceived on striking a mass of jelly, and very significant of the existence of the cyst. Owing to the pressure the increasing tumor may exert on adjacent structures, we observe in some cases dry cough; palpitation and displacement of the heart; vomiting; possibly slight jaundice.

A fatal issue may at any time ensue by the hydatid tumor bursting into the pleura, or the pericardium, or the peritoneum, and leading to violent inflammation; or by suppuration occurring in the sac, when the symptoms become those of pyæmia. Urticaria has been specially noticed in connection with the rupture of the cysts.

In some countries hydatids are frequent; it is not so in this country. In Iceland these growths developed from the eggs of a tapeworm are so common that they cause one-seventh of the human mortality. In point of diagnosis, it is not generally difficult to detect the presence of hydatids. The disease differs from *abscess of the liver* by the want of febrile action, pain, and great constitutional disturbance; indeed, the latent character of the hydatid tumor becomes of much importance. Its slow growth, too, is very significant. When, as sometimes happens, a hydatid tumor inflames and suppurates, we have nothing to guide us in the differential diagnosis but the history previous to the development of the urgent symptoms. From *cancer of the liver* we distinguish hydatids by the long duration of the case, by the absence of evident cachexia, of local tenderness, and of unevenness of the surface. On the other hand, we have in hydatid tumor the sensation on palpation of elasticity or fluctuation. Under rare circumstances this may happen in medullary cancer, but the rapid growth of the latter and the cachectic symptoms would determine the diagnosis. A *distended gall-bladder* may, like hydatid tumor, be free from pain on pressure, but, unlike this, it is movable, is preceded by attacks of colic, is generally accompanied by deep jaundice, and its situation corresponds to that of the normal gall-bladder.

An *aneurism of the aorta* differs from hydatids in the severe pain the patient suffers, so utterly dissimilar to the absence of pain or to the mere feeling of tension and weight of a hydatid swelling. Then the pulsation and the other physical signs aid us. In *aneurism of the hepatic artery*, which may also present a smooth, throbbing tumor, we are apt to have deep jaundice from compression of the biliary ducts.

Pleuritic effusions have many features in common with those cases of hydatids of the liver in which the growing tumor extends upward

into the chest. All the physical signs of a large effusion may be present, even the dilatation of the thorax and a sense of fluctuation in the intercostal spaces. But the absence of constitutional symptoms, the irregular outline of the dulness on percussion of the hydatid cyst, the great displacement of the heart, and the decided lowering of the upper margin of dulness upon deep inspiration, enable us commonly to detect the real nature of the disease. When the cyst has opened into the lung and the hydatids are being expectorated through the air-passages, the harassing cough, the copious sputum, and the inflammation of the pulmonary tissue which is apt to be occasioned, may cause the affection to be mistaken for pulmonary abscess or phthisis. The surest marks of distinction are furnished by the changed form of the lower part of the thorax, and by finding bile and the hooks of the echinococci in the sputum.

Renal enlargements, such as cysts, hydronephrosis, cancer, are discriminated from hydatids of the liver by the same physical signs that distinguish them from hepatic cancer,—chiefly by the renal tumor having the tympanitic sound of the colon in front of it, by its being but slightly, if at all, affected in position by deep inspiration, and by the direction of its growth. Moreover, the history and an examination of the urine will greatly assist.

Ovarian cysts, unlike hydatids, grow from below upward, are not influenced by deep inspiration, and produce enlargements greatest below and not above the umbilicus; then they have a different outline on percussion from hydatid liver.

But, though we may thus generally distinguish hydatids of the liver from the maladies which have similar symptoms, there are unquestionably cases in which it is extremely difficult to arrive at a satisfactory conclusion. Under these circumstances, an exploratory examination with an aspirator would be proper. We may detect shreds of striated hydatid membrane, and portions of echinococci. Besides, the character of the fluid will assist us in diagnosis. It is as clear and colorless as water, has a neutral reaction, a specific gravity of 1005 to 1011, and contains not a trace of albumin or of urea, but large quantities of chloride of sodium. No other fluid in the human body, whether in health or in disease, presents these peculiarities.

Occasionally portions of the liver are transformed into a mass consisting of connective-tissue stroma and numerous cells filled with a gelatinous substance. The disorder looks like alveolar carcinoma, but it is really *multilocular hydatids*, or echinococcus tumors. The centre of the mass suppurates, but even this does not diminish the resistance of the hepatic tumor; nor is fluctuation, save in the rarest

instances, perceptible. Elevations may be found, such as we observe in carcinoma and syphiloma: indeed, the affection is not to be distinguished with any certainty from either, except it be by the history and the attending constitutional symptoms. No jaundice usually accompanies the hard hepatic swelling; but in cases in which the bile-ducts are obstructed we meet with jaundice without dyspeptic symptoms or previous paroxysms of pain, and usually without enlargement of the gall-bladder. In cases with icterus, unlike what we find in syphilis or in cancer, there is complete decoloration of the fæces.[1]

Let us now, in concluding the review of the hepatic maladies which are attended with decided increase of the size of the organ, briefly contrast their most important manifestations. We have found that, as regards the enlargement, they differ materially. Simple congestion, chronic inflammation, fatty liver, hypertrophic cirrhosis, do not attain nearly the volume of cancer, of hydatids, of abscess, of waxy disease of the liver. The three affections first mentioned differ, moreover, from all the others, except the waxy liver, by presenting a uniform and not an irregularly shaped swelling or an uneven outline of the percussion dulness.

Concerning the symptoms, we observe that, although these hepatic disorders all agree in not being characterized by *jaundice*, yet this sign is more commonly present and more distinct in some than in others. In hydatids, and in the syphilitic liver, there is no yellow hue of the skin or of the conjunctiva; so, too, as a rule, in waxy liver. In fatty liver and in abscess it is, on the whole, most frequently wanting. The same may perhaps be said of cancer, yet not infrequently there is deep jaundice in this malady. In chronic congestion, in chronic inflammation, and in hypertrophic cirrhosis, we ordinarily find jaundice, though it may be but a slight yellow tinge of the skin and the eye. With reference to *dropsy*, we are not apt to encounter it in any of the hepatic affections under consideration except cancer, and waxy disease when more than the liver is implicated. It is in these two complaints, also, that the most obvious signs of a cachexia are met with; while in abscess we find fever, and, perhaps, the greatest constitutional disturbance.

As regards *pain*, the fatty liver, hydatids, simple hypertrophy, and the waxy liver are painless; the most painful are cancer, acute cholecystitis, and abscess. Pain is a less prominent symptom in syphilis of the liver and hypertrophic cirrhosis.

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[1] See the cases of Friedreich and of Niemeyer, referred to in Niemeyer's Practice of Medicine.

Chronic Diseases attended with Decreased Size of the Liver, and with Abdominal Dropsy.

Cirrhosis.—Increase of connective tissue producing hardening of the organ is the underlying change in all forms of cirrhosis of the liver. The atrophic form with its granulations of various size, the "hobnail liver," is the most common form, and alcohol the common cause. But this cause does not explain all cases: in some, the malady is connected with syphilis; in others, with malaria; in others, with anthracosis; in others, with infective diseases; in others, again, it cannot be attributed to any known agency, and has been stated to be due to microbic infection. Again, there may be granular livers in which the fibroid tissue is formed between the lobules, and which never contract,—an interstitial hepatitis, or hypertrophic cirrhosis. Cirrhosis is essentially a disease of middle-aged men; it is far less common in women, and rare in children.[1]

In the first stage of cirrhosis, the ordinary or alcoholic cirrhosis, as it is sometimes termed, the organ is somewhat increased in size; then the bulk becomes lessened. It is, however, doubtful whether the stage of enlargement invariably precedes that of shrinking: the process of reduction constitutes not infrequently the first change. But, without entering into this question, we may state that there are no symptoms by which we can recognize the disease at an early period, for the symptoms at first are the same as those of chronic congestion,—dull pain, perhaps tenderness at the hypochondrium and pain referred to the shoulder, disordered digestion, and a sallow or a slightly jaundiced hue of the skin. Nor can we say, even after the stage of contraction is fairly developed,—and it may never reach the point of the hobnail liver being really small,—that the diagnosis of the affection is always possible. It may rest on no stronger grounds than finding in a person who is known to be a spirit-drinker, "a tippler," an intractable ascites, without obvious cause for the dropsy. The dropsy, due to the obstruction of the portal circulation, consists throughout strikingly of ascites; as it increases, œdema of the legs may be developed, and passing albuminuria, from pressure on the renal veins, or beginning cirrhosis of the kidney.

Besides the dropsy, the other clinical features of the malady are not very marked. The most significant signs consist in the diminution of the percussion dulness in the hepatic region, and the detection, by the touch, of firm, irregular granulations on the margin and under

[1] See, however, cases by Howard, Transact. Assoc. Amer. Phys., 1887.

surface of the liver. But both these signs are very difficult to discern, on account of the distention of the abdomen with fluid, and the displacement of the liver this may occasion. In fact, it is often only after the performance of paracentesis that the abdominal walls will permit us to judge with any accuracy of the shrinking and altered state of the organ. This is especially true with reference to palpation; as regards percussion, it may be possible, even when the abdomen is still full of dropsical effusion, to detect the lessened extent of hepatic dulness.

Irrespective of these phenomena, we find at times other manifestations of disease which assist us in the diagnosis of cirrhosis. They are enlargement of the spleen; dilatation of the veins of the abdomen; gastric and intestinal derangements; hemorrhoids; marked loss of flesh and strength; jaundice coming and going, never very striking; a decidedly cachectic appearance, with sunken features; and hemorrhages from the nose and mouth, or from the stomach or intestines, or into internal cavities. Hæmatemesis in an alcoholic must always arouse suspicion. The increase in size of the spleen is far from constant, and rarely reaches a considerable extent. There is often pain over the region of the liver and spleen, and occasional attacks of perihepatitis and of peritonitis occur. The dilatation of the abdominal veins is not perceived until an advanced stage of the disease, and is sometimes connected with a peculiar vascular net-work, stretching from the umbilicus upward and downward, and, as Sappey[1] was the first to describe, with a decided enlargement of the epigastric and mammary veins, the blood flowing through the former in a reversed direction from what it does in health,—namely, not towards the liver, but from it to the veins of the abdominal wall, and thence to the vena cava. Other external veins share in the enlargement; the veins of the legs may be varicose, and the venous twigs on the cheeks become developed. In some cases an irregular but moderate fever not exceeding 102.5° is also noticed; very generally there is none.

Another symptom to which I have had my attention strongly directed is the presence of small amounts of sugar in the urine. Thus, in two cases which I saw with Dr. Simpson, Trommer's test readily detected sugar in the urine. In the one case the secretion was scanty; in the other it was abundant. One had lasted for several years, and was slowly developing; the other had existed about sixteen months, and was rapidly progressing.

Cerebral symptoms due to a toxic cause sometimes appear. They

[1] Bulletin de l'Académie de Médecine, tome xxiv.

show themselves frequently in a delirium of mild type, attended with confusion of persons and places. The delirium is often like that of uræmia, but there is nothing in the urine to account for it. It may not show itself until towards the end of the disease: on the other hand, it may be of long duration. In a case I saw with Dr. Lloyd, it lasted four months. Coma and convulsions also occur occasionally.

The gastric and intestinal derangements, the result of a congested or inflamed mucous membrane, are rarely wanting: they manifest themselves by failing appetite, impaired digestion, both gastric and intestinal, morning sickness, flatulency and constipation, or the frequent voiding of pale-colored stools or attacks of diarrhœa. The jaundice very rarely attains a high degree. It shows itself usually in a yellowish tinge of the skin and conjunctiva; but even this hue is often absent, and we find the pale skin and pearly eye of anæmia.

Yet not one of these symptoms is really characteristic: they become so only when viewed in connection with the dropsy, with the local signs in the hepatic region, with the history of the case, and with the absence of any organic disease of the stomach or the intestine, which might explain them. Then the age of the patient, generally above thirty-five years, and his habits, must be taken into account. The cirrhosis of young children is generally due to inherited syphilis. Gout seems to predispose to the disease. Murchison tells us that the condition of the liver which develops gout renders it liable to suffer from alcohol. Cirrhosis of the liver often becomes associated with acute tuberculosis. At times cirrhosis runs a rapid course.[1]

There is a form of cirrhosis due to infection. It has been described as [illegible]. It is attended with irregular fever of remittent or intermittent type, with marked enlargement of the spleen and splenic pain, with nephritis in the urine, with greatly lessened renal excretion of urea, but much sweating, with slight jaundice and various discoloration of the skin of the liver. The infection occurs, probably, through the intestine, and from the liver spreads along the hepatic veins to the vena cava and may ultimately infect the arterial system, giving rise to infectious nephritis and purulent meningitis. A similar disease is met with in children, a cirrhosis with jaundice after infectious maladies, such as scarlet fever or measles.

Another form of cirrhosis, if it be a form and not a separate disease, by malarial fever, has been described.—[illegible]

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[1] [illegible] Arch. de Méd., Paris, 1882.

[illegible] Med. Ass. [illegible]

or "interstitial hepatitis," or cirrhotic enlargement. It may be found in alcoholics, but often shows itself without recognizable cause. It is frequently noticed in young persons. It has much the same symptoms as atrophic cirrhosis, and is undistinguishable, except by the increased percussion dulness it presents, and by the signs of enlarged liver being usually attended with more decided and much more constant jaundice and greater tendency to protracted fever and to peritonitis. Pain over the liver and spleen, due perhaps to attacks of perihepatitis, is not uncommon. Ascites is absent or slight. The edge of the enlarged liver is hard and not irregular; the gall-bladder is not distended. A peculiar mawkish odor of the breath has been spoken of as present.[1] Dilatation of the abdominal veins is generally absent.

The disease usually begins with the signs of congestion, acute or chronic, with jaundice, and with some pain in the right hypochondrium, and lasts for years, terminating in a slow cachexia; at the end there are marked jaundice and diarrhœa, and the patient sinks into a typhoid state. Ascites may be, as already indicated, wanting throughout; or, as is more usual, it comes on late in the malady. The disease is, in my experience, not infrequently complicated with a fatty liver, forming "a fibro-fatty liver." In some instances of hypertrophic cirrhosis there is organic disease of the heart. The infectious nature of hypertrophic cirrhosis has been often affirmed.

Cirrhosis of the liver due to *malarial infection* is also associated with enlargement, at times very great. It presents, moreover, a persistent chronic jaundice, which may last for years, and is combined with marked enlargement of the spleen and manifestations of the malarial poisoning. Bleeding from the nose, gums, and intestines is frequent; dropsy and distention of the abdominal veins are absent.[2] The disease I believe to be a very rare one.

Let us now look at the distinction between ordinary cirrhosis and some of the maladies which resemble it; and first let us compare its traits with those of other *hepatic affections*. From diseases of the liver attended with enlargement, such as waxy liver, fatty liver, and chronic congestion, fully developed cirrhosis is discriminated by the presence of ascites and the other signs of seriously obstructed portal circulation, by the diminished, or certainly not augmented, size of the organ, and by the different history of the disorder. From *hydatids* of the liver we diagnosticate cirrhosis by the irregularity of outline of

[1] Duckworth, St. Bartholomew's Hospital Reports, 1874.

[2] Lancereaux, quoted in Sajous's Annual, 1888, p. 335.

the enlarged liver in the former complaint, by the sense of fluctuation, and by the comparatively unimpaired general nutrition of the body. *Cancer* of the liver is unlike cirrhosis in the distinctness and size of the protuberances, in the obvious hepatic enlargement, in the less marked ascites, and in the normal size of the spleen. But when a cirrhosed liver is associated with syphilitic nodules, or when its volume is augmented by waxy infiltration, the discrimination from cancer becomes a matter of extreme difficulty; indeed, it may be impossible to avoid erroneous conclusions. Hypertrophic cirrhosis may also be very difficult to distinguish from cancer, except by the history of alcoholic dyspepsia, and, though large and nodulated, the liver is rarely so tender, and the nodules, if they can be felt at all, are small, and ascites is not, as in cancer, a frequent symptom. *Syphilitic hepatitis* cannot be distinguished from hypertrophic cirrhosis, save by the history of the case and feeling the gummata. In some instances there is distinct fever, which subsides under iodide of potassium. The general health may be but little disturbed. In the interstitial hepatitis due to inherited syphilis, enlargement of the liver and jaundice occur.

We shall now consider and compare the clinical traits of some diseases of the liver producing, like ordinary cirrhosis, atrophy of the organ.

As the result of repeated attacks of perihepatitis, we find great thickening of the capsule, with fibrous bands passing into the interior of the organ, and some atrophy. This condition, described as *simple induration of the liver*, is met with chiefly in connection with constitutional syphilis, though it is also seen following a right-sided pleurisy and diseases of parts contiguous to the liver, producing inflammation which spreads to it. The affection is not to be distinguished from true cirrhosis, except by the causing elements, particularly by the syphilitic history, and by the absence of the habit of spirit-drinking; the greater and more persistent pain and tenderness in the hepatic region are of significance; sometimes there is coexisting heart disease.

Red atrophy is a pathological state rather than a recognizable disease. The diminished hepatic dulness is not preceded by alcoholic dyspepsia or valve disease, but is met with in those with a history of dysentery or of ulceration of the intestine. It may be also due to obstinate malaria, and the liver is then at first large and red.

An inflammation of the portal vein, with coagula forming in it, may occasion the same manifestations of deranged abdominal circulation, the same or greater tumefaction of the spleen and decrease of the liver, as cirrhosis. And what complicates the diagnosis very much

is, that cirrhosis is the chief disease that leads to thrombosis of the portal vein. Indeed, we cannot, under any circumstances, positively discriminate this affection from cirrhosis. Still, we are sometimes enabled to distinguish the venous disorder by laying stress on the sudden development of the symptoms, especially of the violent engorgement of the portal system; and by noting the rapidity with which the ascites returns after paracentesis, the rapid swelling of the spleen, the copious gastric or intestinal hemorrhage, the severe vomiting and diarrhœa, the great enlargement of the abdominal veins, and, when not too soon fatal, the marked emaciation. Other causes than inflammation of the coats of the vein, whether simple or infective, may produce coagulation. We may have thrombosis as the result of disease of the liver structure, in cirrhosis, or cancer, or syphilis; or of compression by enlarged cancerous or tubercular glands; or in consequence of the perforation of the vein by cancer or by gall-stones, or of sclerotic change of its coats. Compression of the portal vein and of the biliary ducts in the fissures of the liver, from inflammation of the surrounding areolar tissues, may be separated from cirrhosis chiefly by the intense icterus and the complete decoloration of the stools.

Of non-hepatic affections, cirrhosis is most liable to be confounded with *chronic peritonitis;* a mistake rendered the more likely because chronic congestion or even chronic inflammation of the peritoneum may exist as a complication of cirrhosis. But, even when no such complication is present, the diagnosis may be difficult. It rests chiefly upon the greater and more extended tenderness of the abdomen in peritonitis, the febrile signs, the absence of splenic enlargement and of dilated veins, the usually unchanged, or certainly not jaundiced, hue of the skin, the association with signs of disease in other viscera, especially of the lungs,—for chronic peritonitis is generally tubercular.

Under rare circumstances, *cancer of the stomach* may simulate cirrhosis. I had some years since a case under my charge at the Pennsylvania Hospital, in which, with very slight digestive symptoms, and without discernible epigastric tumor, considerable ascites and effusion into the left pleural cavity existed. Owing to this effusion, the state of the spleen could not be accurately ascertained. There was some fulness of the abdominal veins, and the hepatic percussion dulness did not extend entirely to the margin of the ribs. Bile-pigment was present in the urine, the bowels were loose, and progressive emaciation ensued. The man had been very intemperate, and his case might certainly have been selected as an illustration of cirrhosis; yet at the autopsy the liver, though small, rather hard, and deeply con-

gested, was not cirrhotic, and a cancer involving the whole stomach, except the pylorus, was found.[1]

Chronic Atrophy of the Liver.—Although cirrhosis is the most frequent it is not the sole cause of dwindling of the liver. We have just spoken of its diminution in consequence of obstruction of the trunk of the portal vein, as well as of other causes; but besides these causes we find some, such as a decrease of the organ from long-continued closure of the common duct, or its atrophy in old age, or in connection with grave disease of the heart or lungs obstructing the circulation and causing persistent hyperæmia of the liver, or as an accompaniment of chronic disease of the intestine. The first of these morbid states is mainly discriminated by the deep jaundice, without marked ascites and enlarged abdominal veins; the second, by the absence of any important symptoms referable to the liver and associated with the diminished hepatic dulness; the third, by the history of the case, the physical signs of cardiac or pulmonary difficulty, and the more general dropsy. The fourth form has been mentioned under red atrophy. We may sometimes suspect the cause of the shrinkage of the organ from the persistent and intractable diarrhœa and disturbance of the stomach. But there is no cause of simple atrophy of the liver so common as thrombosis of the portal vein.

SECTION IV.

ABDOMINAL ENLARGEMENT.

In describing the causes of abdominal enlargement, I shall view them as they occasion a general and uniform or a more circumscribed and partial swelling.

General Abdominal Enlargement.

Ascites.—The collection of serous fluid in the peritoneal sac, or ascites, may form part of a general dropsy, and be dependent upon an organic disease of the kidneys or of the thoracic viscera; or the accumulation of liquid may be confined to, or occupy principally, the abdomen. In either case the local signs are much the same. They are: enlargement of the belly; a dull sound on percussion, due to the presence of liquid; and the sense of fluctuation imparted to the hand on one side of the abdomen by a wave of fluid put into motion by a tap on the other side.

[1] For a fuller report of this case, see Proceedings of the Pathological Society, Amer. Journ. Med. Sci., vol. lii., 1866.

As regards the former of these signs, it is uniform and progressive, and is generally very evident; although, of course, when the quantity of liquid is small, enlargement of the abdomen may escape detection. The percussion dulness is most readily perceived at the lower portion of the abdomen, where the fluid gravitates. The bowels float usually to the upper part of the liquid, and at this spot their tympanitic resonance may be distinctly discerned. When the patient is in the erect position, the intestinal percussion note is commonly discoverable in the epigastric and umbilical regions. If he be placed upon his back, the tympanitic sound is found to extend lower than the umbilical region, while dulness will be elicited in the hypogastric region and the flanks. If he be placed upon his side, the flank which is uppermost becomes resonant. This alteration of the level of the fluid with the change of position is thus a significant sign, and always happens except when the effusion is encysted; it is detected without difficulty, save where great flatulent distention of the bowels or impaction of fæces accompanies the accumulation of liquid.

Ordinarily, the fluctuation wave felt by the hand is easily discerned. It is obscured by thickening of the abdominal walls from œdema, or from the accumulation of fat in the subcutaneous tissues; it is, moreover, indistinct if adhesions circumscribe the fluid in the peritoneum. The amount of albumin in the fluid rises with the ascites and its duration. For all practical applications the specific gravity determines the proportion of albumin, and the urinometer may be employed for the purpose.

There are no means of distinguishing the character of the fluid except by direct observation. Chylous ascites has been not infrequently found associated with tubercle,[1] or cancer of the peritoneum. It has also been met with in filariasis and in rupture of the thoracic duct. A hemorrhagic fluid indicates cancer or tubercle of the peritoneum, though it is occasionally seen in cirrhosis.

The other symptoms often found in ascites, such as a pushing upward of the liver, spleen, and stomach, embarrassed breathing compression of the lungs, and digestive disturbances, present nothing characteristic. But we insist on this: that a diagnosis of ascites is only half a diagnosis, and that we should in every instance endeavor to ascertain the cause of the collection of fluid in the peritoneal sac. The morbid states with which dropsy in the peritoneum is liable to be confounded are chiefly:

[1] Busey, Amer. Journ. Med. Sci., Dec. 1889.

Ovarian Dropsy;
Chronic Peritonitis;
Distention of the Bladder;
Gravid Uterus;
Chronic Tympanites.

Ovarian Dropsy.—It is not until an ovarian cyst rises above the brim of the pelvis that it occasions a swelling marked enough to be mistaken for abdominal dropsy. Supposing that it has led to considerable enlargement of the belly, we are yet able to discriminate between the two disorders by attention to the physical signs of the history of the case.

As regards the former, we perceive these differences: the sound on percussion over an ovarian cyst is dull in the umbilical and hypogastric regions, while at the sides the tympanitic resonance of the intestines may be obtained. Moreover, the dulness in ovarian dropsy does not change its position in different postures; and, like all ovarian tumors, the ovarian dropsy causes a projection in the centre of the abdomen, not a flattening there and a bulging of the flanks, as is common in ascites. Bacelli[1] states that in ascites there is a deep tympanitic sound during percussion in the region of the intestines, while an ovarian cyst presents dulness on the side in which the cyst has its origin, and a tympanitic sound on percussion on the other. In ascites, vaginal and rectal touch detect fluctuation at once, and the uterus is normal in size and in mobility, sometimes it is prolapsed; in ovarian dropsy, fluctuation is less distinct, and may not be found at all, and the uterus is generally displaced behind the cyst.

The fluctuation from an ovarian cyst is unequal at different parts of the distended abdomen. When the effused fluid is free in the peritoneal cavity, fluctuation may be perceived beyond the line of dulness as the fluid is thrown in waves among the intestines; but when it is confined within a cyst, fluctuation cannot be perceived beyond the cyst walls: hence the outline of the cyst as obtained by percussion, and that of the area within which fluctuation is perceived, must be the same. It should be remembered, however, that fluctuation in an ovarian cyst may escape detection on account of the great thickness of the cyst walls, or of the unusual tenseness of the cyst, or of the great density of the fluid, or of the small amount of fluid in each cyst. In ovarian cyst there is, for the most part, impairment of the general health, and the color of the face is that of cachexia.

When there is ascites complicating an ovarian tumor, the diagnosis

[1] Wien. Med. Wochensch., April, 1890.

is very difficult. Finding the fluctuation unequal, and an irregular outline of the ovarian growth, may aid us; but a preliminary tapping, though now mostly condemned by gynæcologists, may be necessary to arrive at an opinion. The specific gravity of the fluid of ovarian cysts is 1020 to 1025, thus considerably higher than of ascitic fluid, which is generally about 1010. Entire reliance cannot be placed on the chemical character of the fluid, since the rule that paralbumin is significant of ovarian fluids and fibrin of serous fluids has many exceptions. Spencer Wells[1] accepts the presence of the "granular cell," as shown by Drysdale and W. L. Atlee,[2] to be characteristic of ovarian fluid. This granular cell is generally round, sometimes oval, varies in diameter from one five-thousandth to one two-thousandth of an inch, is transparent, is much smaller and far less opaque than the compound granular cell of inflammation, and contains a number of fine granules which become more distinct on the addition of acetic acid, and nearly transparent under ether; there is no nucleus.[3]

In uncomplicated cases, the history assists us greatly in reaching a correct diagnosis. In ovarian dropsy, we can, as a rule, make out that the distention of the abdomen has begun at its lower portion on one side, and has spread upward. Again, we do not find those signs of disease of the liver, heart, kidneys, or spleen which are so apt to coexist with ascites, or that the swelling is reduced by the use of hydragogue cathartics and diuretics, as in the latter complaint.

Attention to the history and progress of the complaint is especially valuable in the class of cases in which the physical signs of ascites are modified by the intestines not being able to float to the surface of the fluid in the peritoneal cavity, in consequence of adhesions to one another, or of a diseased omentum, or in which the fluid has been limited in sacs by inflammatory adhesions. On the other hand, an ovarian cyst may contain air, either from a communication with the intestine, or after tapping and decomposition of the contained fluid, and percussion would then give a clear note in front and a dull note below; succussion, too, has been noticed. In the diagnosis between encysted dropsy of the peritoneum and an ovarian cyst, if we obtain, by tapping, a spring-water fluid, it points to cyst of the broad ligament.

Chronic Peritonitis.—We find chronic peritonitis as the result of an acute attack, or in connection with cirrhosis of the liver, with dila-

[1] Brit. Med. Journ., June, 1878.

[2] Ovarian Tumors.

[3] See Transactions of the Pathological Society of Philadelphia, vol. vii., 1877; American Journal of Obstetrics, vol. xii., 1879; also Gynæcological Transactions, 1883.

tation of the colon, with chronic dysentery, or with interstitial nephritis. But usually the peritonitis is either tubercular or cancerous.

Tubercular peritonitis generally occurs in those who have tubercles in the lungs or enlarged caseous glands; and when such patients complain of abdominal pain and uneasiness, of soreness to the touch, of nausea and vomiting, of diarrhœa alternating with constipation, and of losing flesh and strength; when the tender abdomen is tense, resistant, much distended, in part with liquid, but especially with wind, and exhibits on its exterior the tracings of the convolutions of the intestines; when in addition there is œdema of the lower limbs, with fever, irregular, at times high, at times almost ceasing, and a growing cachexia,—we can hardly be wrong in presuming the signs of chronic peritoneal inflammation to be owing to the presence of tubercle. Even when disease of the lungs is absent, or is not well defined, we shall generally be correct, if the abdominal symptoms mentioned exist, and there are repeated attacks of acute or subacute peritonitis, in determining the peritoneal affection to be tubercular. Signs of great significance are the presence of nodules in the rectum and in the sacro-uterine ligaments, and of inflammation around the Fallopian tube. In some instances the disorder develops with rapidity, and has the aspect of an acute complaint. On the other hand it may be latent. The tumefaction of the belly may be so great as to simulate an abdominal tumor.[1] The disease is often mistaken for ovarian disease.

A *cancer of the peritoneum* gives rise to many of the same phenomena as tuberculous disease. But the affection is far less common, and there is this difference: the malady usually happens consecutively to an external or an internal cancer, and scarcely ever save in persons advanced in years; there is little or no fever, or, indeed, a subnormal temperature, and neither diarrhœa nor profuse sweats. Pain, on the other hand, or at least attacks of spontaneous pain, are more frequent; the lymphatic glands enlarge; and, as the omentum is the most common seat of the cancerous growth, we can generally detect a tumor stretching across the upper portion of the abdomen. The morbid mass is unequal, and usually discovered readily, except where separated by fluid from the abdominal parietes. There are often nodules in the neighborhood of the umbilicus and enlarged inguinal glands; a peritoneal friction-sound is heard. Hemorrhage into the abdominal cavity or the effusion of bloody serum occurs in cancerous as it does in tubercular peritonitis. In cancerous peritonitis the ascitic

[1] See case in Liverpool Hospital Reports, 1868.

fluid has a turbid gray look. In the sediment that forms there is a rich cell-growth with many red blood-corpuscles. The cells are for the most part peculiar, large, swollen, nucleated cells;[1] many are multinuclear cells. In primary cancer of the peritoneum, or that following *cancer of the retroperitoneal glands*, the diagnosis is very obscure, unless the tumors are marked. The cancerous malady pursues a slowly progressive course, lasting months; but it may develop as an acute miliary disease. Retroperitoneal tumors may be readily mistaken for diseases of the liver. They may occasion jaundice from pressure on the common duct. The fact that they do not move with the acts of breathing, as well as that there is often a line of resonance between the dulness they occasion and the liver dulness, is a point of value in diagnosis.[2]

Distention of the Bladder.—This may give rise to a sense of fluctuation and to very marked abdominal enlargement; so marked, indeed, that patients have been tapped, under the supposition that they were laboring under dropsy of the abdomen. But when the bladder is so much distended as to simulate ascites, there is more or less tenderness on pressure over the seat of the obvious swelling; which, moreover, presents a rounded outline of dulness on percussion. Again, we have the history either of retention or of apparent incontinence of urine.[3] But, to avoid all possible chance of error, in any case of doubt a catheter should be introduced into the bladder. This mode of procedure, it may here be mentioned, is the one which leads most speedily and decisively to a true appreciation of the abnormal phenomena in those rare cases of anasarca which are produced by distention of the bladder, and of which Trousseau has recorded several.

The Gravid Uterus.—A gravid womb is readily distinguished from abdominal dropsy by the peculiar form of the dulness on percussion, its steady and uniform increase corresponding to the enlargement of the womb, the absence of fluctuation, the detection of the sounds of the fœtal heart, the alteration in the color and appearance of the mammary areola, and the production of movements in the womb on making an examination per vaginam.

Chronic Tympanites.—Great prominence of the abdomen, due to flatulent distention of the bowels, is, if at all persistent, very apt to

[1] Runeberg, Deutsches Archiv f. klin. Med., Sept. 1883; also Coe, New York Med. Journ., July, 1888.

[2] Vander Veer, Amer. Journ. Med. Sci., Jan. 1892.

[3] In a case recorded by Watson, in his Lectures on the Practice of Physic, although the bladder was enormously distended, large quantities of urine were constantly passing from the patient.

be mistaken for ascites. But the large abdomen yields not a dull, but everywhere a tympanitic sound, and there is no fluctuation. Then the history of the case and the attending symptoms throw light upon the nature of the ailment. Many persons suffering from chronic tympanites have all the signs of weak gastric or intestinal digestion; in others there is hysteria.

Among soldiers this chronic tympanites—owing, perhaps, in many cases to the character of their diet and consequent digestive disturbances—is far from being an uncommon disorder, and may be a very obstinate one. It gives rise to abdominal enlargement, which is constantly mistaken for dropsy, but which does not yield a sense of fluctuation, or return on percussion any other than a well-marked tympanitic sound. The distention produces, moreover, an inability to take active exercise, sensations of cutting pain under the ribs, and palpitation of the heart; pressure on the abdomen occasions much discomfort; the soldiers, therefore, walk with their clothes unbuttoned, and find it very irksome to wear their belts. They are sometimes troubled by indigestion, and feel particularly uncomfortable after meals; or the symptoms of indigestion, although they may have been present at the beginning of the complaint, disappear, but the swelling of the abdomen persists for many months. According to my experience, the ailment is always gradual in its development.

Besides the complaints just reviewed, which are those most commonly confounded with ascites, there are a few very rare disorders which might be mistaken for collections of fluid in the peritoneal sac. They are dropsy of the womb; dropsy of the Fallopian tubes; dropsy of the omentum; very large serous cysts in the kidney; hydatids of the liver, of size so great as to lead to general abdominal distention; and a dilatation of the stomach so extensive that the viscus occupies almost the whole abdomen. With reference to the latter affection we may distinguish it from ascites by the history of the case and the vomiting and other marked gastric symptoms, by the extended tympanitic percussion note, by the indistinct fluctuation, which is not noticed except over the most dependent part of the organ, by the splashing or the metallic or amphoric sounds which are perceived when its contents are agitated, by the length to which the stomach-tube can be introduced, and by the chemical examination of the gastric contents. The other maladies mentioned can be separated only by taking into account their history and progress, and by laying stress upon the absence of those morbid states which generally cause ascites, and upon the occurrence of special phenomena which point to the structures implicated.

Partial Abdominal Enlargement.

Abdominal Tumors.—Even at the risk of repetition, it is for clinical purposes a matter of convenience to point out connectedly the relations an abdominal swelling bears to the normal structures of the abdominal cavity, and to consider, moreover, the swelling as constituting the starting-point of our diagnosis.

Let us first examine into the meaning of an abdominal tumefaction occupying solely or principally one region of the abdomen.

Right Hypochondrium.—The most usual cause of a tumor in this region is an enlargement of the liver. Sometimes a tumor which is in the lower part of the right hypochondrium, or proceeds from the termination of this region, is simply a displaced liver, or an affection of the gall-bladder. In the first instance, the recognition of the disorder —such as a pleuritic effusion—which has given rise to the displacement; in the second, the history of the case, the shape of the swelling, and the symptoms attending it,—will give us an insight into its cause. Again, a tumor in the parts mentioned may be due to an enlarged kidney, cancerous or cystic, or especially hydronephrosis. Careful examinations of the urine and the history of the case furnish the most certain means of discrimination. Then we must also bear in mind that all enlarged kidneys displace the bowel in a particular manner; they press it forward, and the dulness over the tumor is largely mixed with a tympanitic sound, or the dulness is, indeed, not very appreciable.

Left Hypochondrium.—The most usual tumors in this region are produced by enlargement of the spleen. An increase in size of this viscus, if acute, is generally owing to toxæmias, acute fevers, and bacterial infection, as pyæmia, puerperal fever, acute tuberculosis, scarlet fever, typhoid fever, relapsing fever, or the malarial fevers. The cause of the swelling is disclosed by the history of the case and by the accompanying symptoms.

Inflammation of the spleen is an affection very difficult to recognize. The most trustworthy symptoms are: pain in the left hypochondrium, radiating as far as the left shoulder, and augmented by pressure by coughing, and by a deep inspiration; nausea and vomiting; fever having irregular fits of exacerbation; sometimes delirium, dry cough, and a sense of suffocation. The extent of the splenic percussion dulness is decidedly increased, and, when we are sure that the spleen is not displaced, the suddenly widened area of dulness forms an important element in the diagnosis. Splenitis is rarely primary, is generally from pyæmia and from infarcts. It is often observed to be

connected with emboli from endocarditis, and, these being wafted also to the kidneys, albumin and blood are found in the urine. When suppuration in the spleen ensues, of which the general cause is infective endocarditis, the fever may assume a hectic character and the patient lose flesh rapidly, while the spleen increases in size. But there is no certainty in these signs, nor, indeed, in any of the signs of splenic abscess; this may be latent and suddenly rupture into the abdominal cavity or the stomach. Then there may be abscesses around the spleen with manifestations similar to those in its substance, or to pyopneumothorax.[1] An acute enlargement of the spleen may also be owing to hemorrhage from injury.

Chronic enlargement of the spleen may be caused by hypertrophy, by waxy disease, by leukæmia and lymphadenoma, by splenic anæmia, by a malignant growth, by hydatids, by syphilitic tumor, by congenital syphilis, and by structural changes from malaria. There are scarcely any symptoms characteristic of these states, except the alteration the blood undergoes, evinced often by a diminution of the red globules and an increase of the white. But this, as we shall find in studying the blood, depends very much upon the special disease. Waxy hue of the face, dropsy, bleeding from the nose, from the stomach, or from the intestinal canal, and digestive disturbances, though far from infrequent, are also not constant signs. Death even may result, as from rupture of varices of the enlarged viscus, without any other manifestations of a lesion than increased size of the organ.[2] When enlargement of the spleen has reached a certain point, the organ curves into the hypogastric and right iliac regions, and a notch or notches may be felt on its anterior and inner surfaces.[3] This sign may be very valuable in distinguishing the enlarged organ from cancer of the kidney, for which it has been mistaken.[4] In some instances enlargement of the spleen is hereditary.[5]

Having determined the persistent swelling to be due to the abnormal size of the spleen, we must next endeavor to ascertain the cause of it. The history of the case and the blood examinations are the main elements in diagnosis.

A fulness projecting from the left hypochondrium towards the umbilical or lumbar region may be owing to *fecal accumulations* in the colon. Although these fecal accumulations do not occur so often

[1] Zuber, Revue de Médecine, Nov. 1882.

[2] Traube, Virchow's Archiv, 1869.

[3] Fagge, Guy's Hosp. Rep., 1868.

[4] Lancet, July, 1873.

[5] Wilson and Stanley, Clin. Soc. Trans., 1893.

in or near either hypochondrium as they do in the iliac regions, yet they are not very uncommon, and we should be on our guard against confounding them with organic disease, whether of the stomach, spleen, liver, kidneys, peritoneum, or ovary. Their irregular outline, their doughy consistence and painlessness, and attention to the history of the case and to the accompanying disorder of the digestive functions, will generally enable us to detect the true nature of the swelling. But we must not lay too much stress on the non-existence of constipation, for sometimes great irritability of the bowels or persistent diarrhœa is kept up by a large collection of fecal matter in the colon, and an irritative fever superadded gives a strong resemblance to typhoid.[1] Repeated attacks of colicky pains and soreness to the touch are not unusual in cases of extensive fecal accumulation, and jaundice and anæmia have been also noticed. Besides looseness and mucus, the stools are apt to show small, hard, fecal masses, of leaden hue. In cases of doubt, laxatives, especially castor oil, should be employed before any opinion is given, and with the voiding of large masses of fæces the tumor and the attending symptoms may disappear.

As regards swellings of any kind situated in either hypochondrium, or in fact at any portion of the upper third of the abdomen, we should always observe whether they are affected by the act of respiration. This is a valuable sign, for if the morbid mass move in consequence of the depression of the diaphragm, it is because structures are involved, such as the stomach and transverse colon, the liver or spleen, which admit of some mobility; whereas a tumor that is uninfluenced must appertain to a fixed part,—for instance, to the aorta.

Epigastrium.—The most common cause of an epigastric tumor is cancer of the stomach. The swelling is then associated with the symptoms already described.

But a tumor in this region may be also produced by a *disease of the pancreas.* A swelling occasioned by *fatty degeneration*, or by *uniform simple hardening of the gland*, cannot, as a rule, be discerned at the bedside. In *pancreatic fat necrosis*, the areas of white necrotic tissue are usually also found in the mesentery and in other seats of abdominal fatty tissue. There are no diagnostic signs. In *chronic pancreatitis*, deep-seated epigastric pain and tenderness with colicky attacks, a large quantity of matter like saliva passed by stool, profuse salivation, sugar in the urine, colorless or fatty stools, and jaundice have been observed to attend the appreciable swelling extending across the epigastrium. The association of chronic pancreatitis with

[1] As in a case seen with Dr. Arthur V. Meigs.

diabetes is close. *Suppurative pancreatitis*, as we know from Fitz's analysis, is much more common in women than in men. Though often chronic, it may manifest itself by sharp epigastric pain and vomiting, and is not infrequently attended with chills and irregular fever. It may last weeks or months. A deep-seated resistance over the seat of the pancreas with circumscribed peritonitis, diarrhœa, and slight jaundice are noticed as the case progresses. As regards *cancer*, which can be recognized with more certainty, the most trustworthy symptoms are: a tumor in the epigastric region; pain there or in the back, not increased by the taking of food, but usually augmented by the erect posture; progressive emaciation and debility; an appetite capricious rather than diminished, and in some instances, indeed, a ravenous desire for food; constipation, and at times, but far from invariably, fatty stools, or fat-crystals in abundance in the grayish stools,[1] and profuse salivation. Besides these indications, we commonly find, as the disease advances, obstinate jaundice and occasional vomiting. Many of these phenomena belong also to cancer of the stomach; in truth, we never can be certain of the existence of the pancreatic malady until we have excluded the gastric affection. In a differential diagnosis of this kind, the early presence and habitual occurrence of vomiting after meals, the sour eructations, the hæmatemesis, the want of free hydrochloric acid in the stomach-contents with the presence of lactic acid, and the absence of jaundice, assist us in locating the seat of the disease in the stomach. A *cyst* of the pancreas is distinguished by a smooth round tumor in the epigastrium, slightly movable, and separated by tympanitic percussion resonance from the liver and spleen. When the stomach is inflated, the tumor is found to lie behind and below it. If the cyst be aspirated, an alkaline fluid is obtained which emulsifies fat, transforms starch into glucose, and may digest albumin and fibrin. *Calculous disease* of the pancreas is a very rare affection. There are, in addition to the dull sense of weight at the epigastrium and other symptoms of pancreatic disease,—such as the intermittent presence of sugar in the urine, vomiting, the passage of much undigested muscular fibre, and of fatty stools,—sharp, irregular attacks of colicky pain radiating to the left, due to the passage of calculi; there is no jaundice.[2] Pancreatic calculi may lead to atrophy of the gland and become associated with permanent diabetes.[3]

[1] But collections of fat-crystals, Gerhardt has found, are also detected in the pale stools of icterus without pancreatic disease; when the bile reappears in the stools the crystals are no longer seen.

[2] Fitz, "Diseases of the Pancreas," Allbutt's System of Medicine.

[3] Lichtheim, Berlin. klin. Wochensch., 1894, No. 8.

An epigastric tumor is sometimes simulated by a *contraction of the upper portion of the rectus muscle* on palpation; but the swelling soon subsides, especially if rubbed. Occasionally, however, a tumefaction due to contraction of an abdominal muscle may be of some duration.[1] I have known a contraction of the rectus muscle in a case of gastric cancer occasion so obvious a resistance and swelling that it was looked upon as due to malignant disease of the intestine or of the peritoneum. Moreover, the rigid muscle gave rise to dulness on percussion. But, though the phenomena were for a long period a marked feature of the case, it was observable that the muscle was raised and rigid to a decided degree only in certain positions; at all events, that certain positions gave a distinct outline to the swelling, and that the latter then, like the line of dulness, was regular and straight, evidently corresponding to the contour of the muscle. And this occurs in all instances of contraction of the rectus, no matter with what associated.

The muscular contractions are not always confined to one muscle, or to the whole of one muscle, and when irregular, and particularly when associated with tympanitic distention of the intestine, give rise to most of the so-called "phantom tumors" of the abdomen. These swellings are perplexing, and are constantly mistaken for serious abdominal tumors. The history of the case, the absence of grave constitutional symptoms, the most frequent occurrence of the tumefaction in women, especially in hysterical women, and the usually coexisting constipation, furnish us with valuable signs of distinction. But I believe the use of anæsthetics to be the most important means of diagnosis. I was first led to employ them a number of years ago, in a case which had baffled the skill of several eminent surgeons, one of whom had proposed to the patient an operation as the only means of relief from what was considered an ovarian disease. The patient was thirty-one years of age, a widow, and evidently of highly hysterical temperament. She was very subject to constipation; and the swelling of which she complained was of irregular outline and occupied the centre of the abdomen, extending some distance on each side of the median line. It was hard and resisting to the touch, but, on strong percussion, yielded a tympanitic sound. Whenever it was touched she shrank. Thorough relaxation was produced by the administration of ether; the hand could be pressed almost against the vertebral column, and all signs of the tumor disappeared. A complete recovery took place; and thus terminated a case which had lasted for fully one year. In any instance of phantom tumor I would recommend the use of

[1] Greenhow's cases, Lancet, 1857.

anæsthetics for purposes of diagnosis; nay, they may be most advantageously employed, for similar reasons, in all cases of abdominal swelling in which the rigid state of the abdominal walls interferes with accuracy of investigation. Fitz[1] regards the chronic phantom tumor as identical with idiopathic dilatation of the colon, and the latter as the constant characteristic.

In soldiers we observe at times one or several small movable tumors, yielding a tympanitic sound on percussion, in the epigastric or at the upper part of the umbilical region. They are, probably, small portions of intestine which have been pushed between the fasciculi of a ruptured rectus muscle, similar to umbilical hernia.

Umbilical Region.—Tumors which are found in this region form, as a rule, merely portions of a swelling that is principally seated in the epigastrium or in the hypochondria, such as cancer of the stomach, of the liver, of the pancreas, or of the omentum, and dilatation of the gall-bladder. The only two affections which are apt to occasion a swelling solely, or at least principally, limited to and perceptible in the umbilical region, are tuberculous disease of the mesenteric glands and a movable kidney.

The symptoms of the former malady, or *tabes mesenterica*, are much the same as those of tubercular peritonitis. Indeed, unless the enlarged mesenteric glands can be felt through the abdominal parietes, the discrimination is uncertain. The abdomen is prematurely large, is slightly tender on pressure, and has often a doughy feel; the child loses flesh, the digestion is impaired, the evacuations are frequent, liquid, and offensive. It often presents signs of scrofulous or tubercular disease elsewhere; and under such circumstances we cannot be at a loss in determining the nature of the tumefaction in the umbilical region. The disease is very rare in adults, though it occurs.[2] Its simulation, especially in young women, by *pseudo tabes mesenterica*, has been described in reviewing the affections of the stomach.

When the *kidneys* are not firmly held by their attachments, they become displaced, and are apt to give rise to serious errors in diagnosis. The dislocated organ is perceived under the margin of the ribs on the right flank, or in the umbilical region, and sometimes extends across the median line. The mass is easily moved, may be, by careful and methodical pressure, returned to the renal region, and presents, on palpation and on percussion, the outline of the kidney. The lumbar region yields a tympanitic sound on percussion.

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[1] American Journal of the Medical Sciences, Aug. 1899.

[2] See case reported by Gairdner, Lectures to Practitioners.

and we find less resistance and a slight depression over the usual seat of the organ. But the most certain way of detecting a *movable kidney* is to examine the patient by palpation with both hands, while in the recumbent position with the abdominal walls relaxed, and on deep inspiration the fingers of the right hand will then feel the resistance and the outline of the kidney. There is in some instances sensitiveness over the displaced organ, especially after fatigue or strong pressure; and this occasions the same sensation as when the renal region of the non-affected side is pressed; but we do not find any disturbance of the urinary functions, save, perhaps, frequent urination, nor, in fact, except a disagreeable feeling in walking, does any real inconvenience result from the accident, unless the movable kidney has become painful, or, by compressing the vena cava or portal veins, occasions dropsy. Yet we meet with exceptions to the rule that the disorder gives rise to no decided symptoms. Sometimes dyspepsia, especially nervous dyspepsia, is pronounced, as well as intercostal neuralgia. The stomach is often below the normal level. So-called gastric crises also occur, marked by constipation, a feeling of weight in the abdomen, pain in the sacral region after exertion, throbbing of the abdominal aorta and vomiting, with severe abdominal pain and fever; or there are attacks simulating renal colic. Further, we may find intermitting hydronephrosis.[1] In certain instances the pressure on the bile-ducts from a displaced right kidney gives rise to attacks of hepatic colic followed by jaundice, and leads to the supposition of gallstones.[2] There seems to be a special connection between movable kidney and neurasthenic hysteria, gastric dilatation, enteroptosis, chronic appendicitis of the right side,[3] and membranous enteritis, but the majority of cases are latent, and are only accidentally detected.

The disorder is most apt to occur after violent exertion, or after many pregnancies, or may be due to attacks of congestion of the organ, or to tight lacing. It is rare in men. The right kidney is oftener movable than the left, and it may be felt low down as a movable mass floating near the right iliac fossa. Both kidneys may be displaced.

The affection may be mistaken for any form of abdominal tumor, and if the kidney should have become adherent the diagnosis is uncertain. Generally the disorder can be distinguished by the history of the case, and by the physical phenomena mentioned. To these may be added the comparatively slight dulness or rather the tympanitic

[1] Knight, Lancet, Oct. 1893.

[2] Maclagan and Treves, Lancet, Jan. 6, 1900.

[3] Edebohls, Medical Record, March, 1899.

character of sound elicited, except on very strong percussion, over the seat of the tumor. This is an important fact as regards the discrimination of a movable and *displaced spleen*, in which, as the organ is generally enlarged, there is extended dulness on percussion. Moreover, the history of the splenic disorder, which not uncommonly can be traced to a malarial affection, the usually great tenderness, the nausea, dyspeptic symptoms, and hemorrhagic tendencies which attend the displacement of the spleen, and the notch which can be felt in it, will assist us in our diagnosis. A movable kidney may be simulated by *malignant disease of the colon*.[1]

Yet another of the abdominal organs is occasionally displaced and movable,—the liver. Now, a *movable liver* would be often mistaken for a movable spleen, were it a more common affection. But few well-authenticated cases are on record.[2] In these the peritoneal attachment of the organ had become lax, usually in consequence of pregnancy; in the hepatic region there was a tympanitic sound on percussion; and in the umbilical region and towards the right flank a solid body was discerned, the upper border of which presented a convex outline, the lower border was in the inguinal region. The displaced organ was easily pushed about, and could be replaced in its proper situation. The spleen was found in its usual seat; the symptoms were merely those of weight and uneasiness in the abdomen. The movable or wandering organ may be painful or painless. It has the physical characters of the liver, and the most certain sign is the detection, on palpation, of the notch between the right and the left lobe and of a zone of tympanitic resonance between the swelling and the lung. The diagnosis is, however, always difficult and doubtful. New growths of the kidney, as a case of Legg's proves, are particularly confusing. In most recorded cases autopsies are wanting: and the whole subject is very obscure. The affection is more usual in women than in men, and, besides pregnancy, tight lacing and chronic inflammation of the peritoneum are said to lead to it.

Lumbar Region.—Tumors in this region, or on either flank, are occasioned by some morbid growth of the kidney, or by an abscess in it or its surroundings, or in the psoas muscles. Again, they may be due

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[1] Henry Morris, Lancet, April, 1895.

[2] See Cantani, Ann. Univers di Medicina, 1866; and Meissner's article in Schmidt's Jahrb., 1869, No. 1; also *ibid.*, No. 2, 1871; Blet, Le Foie mobile, Thèse de Paris, 1876; Legg, St. Bartholomew's Hospital Reports, 1877; Arini, Anales del Circulo Méd. Argentino, quoted in Amer. Journ. Med. Sci., July, 1884; H. W. Seager, Brit. Med. Journ., London, 1885, ii.; L. Landau, Deutsche Med. Wochensch., Berlin, 1885, ii.; Richelot, L'Union Médicale, Paris, Aug. 1893.

to fecal accumulations; or, if on the right side, to very considerable increase of the liver; if on the left, to a greatly enlarged spleen. To discriminate between these conditions, we have to determine whether the swelling fluctuates or not; we must also analyze the urine, and inquire minutely into the circumstances preceding and attending the tumefaction. It is thus only that we can attain the necessary data for a diagnosis, which has, indeed, often to be reached by the process of exclusion.

Tumors behind the peritoneum may give rise to a visible prominence in either lumbar region, extending to the upper part of the iliac region. The most common cause of these tumors is *cancer of the lymphatic glands* lying by the sides or in front of the vertebral column. The disease is very difficult of detection. Still, we may suspect its existence if, in a patient who is evidently cachectic and who is steadily losing flesh and strength, we discover, on deep palpation, on one side of the linea alba or in the flank, a tumor which, owing to its being surrounded by intestine, returns a tympanitic percussion sound. In some cases the swelling communicates the beat of the aorta and simulates an aneurism, or it presses on the vena cava and gives rise to enlargement of the abdominal veins and of those of the lower extremities, and to œdema of the legs. The disease may involve the iliac glands and the tumor extend into the pelvis, or it may reach upward to the diaphragm; and, by the cancer spreading to the posterior mediastinum, it may finally open the aorta, producing hemorrhages precisely like those coming from an aneurismal sac.[1]

Iliac Regions.—Tumors in either of these regions may be due to many different causes. They are, as we have elsewhere discussed, principally owing to ovarian affections; to fecal accumulations; to disease of the large intestine, such as intussusception or cancer; and to pelvic abscess. Sometimes they are caused by displacement of the kidney, by enlargement of the spleen, and in women by retrouterine hæmatocele, or by extrauterine pregnancy.

The *ovarian tumors* are, as a rule, distinguished from the other disorders mentioned by their more or less globular form, by their movability from side to side or in an upward direction, by their seeming to spring out of the pelvis, and their evident attachment below, by the displacement of the womb, by the comparatively unimpaired general health, and by their indolent and generally painless nature. These remarks do not apply to the very slight swelling occasioned by ovarian inflammation, for here the tumid spot is often the seat of severe pain. The healthy ovary is not sensitive to the touch. To

[1] Case reported by Haldane, Edinburgh Medical Journal, Aug. 1868.

examine the ovary with exactness, the abdominal muscles must be completely relaxed; the patient is placed in the attitude recommended by Marion Sims,—on her back, with the shoulders supported, the legs drawn up so that the heels are a few inches asunder and the thighs fall easily apart.

As ovarian tumors grow and spread upward they give rise to difficulties in diagnosis, which we have already examined into. We may here again mention the manner in which ovarian may simulate *renal growths*. Stress may be laid on the renal tumor being first detected between the false ribs and the ilium; on the signs in the urine, and on the absence of those changes in the quantity and regularity of the menstrual discharge which are common in ovarian disorders. Moreover, the ovarian growth usually displaces the intestine backward; in the renal growth it is pressed forward and towards the centre of the abdomen; and large tumors of the right kidney ordinarily have the ascending colon on their inner border, while tumors of the left kidney are generally crossed from above downward by the descending colon.

Among the causes of a tumor in either iliac fossa, *retrouterine hæmatocele* has been mentioned. The tumor, commonly of rounded shape, rises above the brim of the pelvis, but is traceable into it. It forms quickly, and an examination through the vagina detects a boggy swelling in Douglas's cul-de-sac, and at times the grating of the blood coagula; faintness and collapse attend its production. Much the same physical phenomena are presented by the swelling due to *pelvic cellulitis*. But the slow way in which the tumor forms, the presence of a hot, puffy, brawn-like condition of the vaginal wall, the usually greater tenderness of the swelling felt through the walls of the vagina, and the feverishness and constitutional symptoms attending the gradual formation of the abscess, are distinguishing marks, except where the contents of the hæmatocele suppurate, when for a differential diagnosis we may have to rely on the history of the case.

Hypogastric Region.—Distention of the bladder and enlargement of the uterus, whether produced by air, by liquid, by a morbid growth, or by pregnancy, are the most usual sources of a swelling in this region. If due to any one of these causes, the outline of the tumor is regular and rounded: and by the aid of the catheter, of explorations through the vagina and the rectum, of the history of the case, and of the attending symptoms, we are generally enabled to arrive at a correct diagnosis.

A tumor in the hypogastrium may also have its origin in splenic enlargement, in diseases of the peritoneum, or in hæmatocele. In the latter case it is apt to be uniform and to extend to the iliac fossæ.

In concluding this sketch of abdominal tumors, we shall briefly glance at those which are likely to occupy more than one region, and sometimes even the whole or greater part, of the abdomen. In rare instances, a cancer of the liver, or hydatids of that organ, or a fibrous tumor of the uterus, or a solid ovarian growth, or an enlarged spleen,[1] or a kidney the pelvis of which has become enormously distended in consequence of obstruction of the ureter, may lead to the formation of a swelling that occupies nearly the entire abdomen. But the most usual cause of so diffuse a tumor is *carcinoma of the peritoneum.* Here there is an irregular tumor, pain, ascites, and, in consequence of the peritonitis set up, fever. Much the same symptoms may be produced by *hydatid disease of the peritoneum*, though there is less fever or none, the swelling may be uniform or even more irregular, the abdominal enlargement greater and painless, and we may be able to detect the hydatid fremitus, and the hooklets in the evacuated fluid.[2] Yet as regards the hydatid thrill we must bear in mind that a similar sensation is obtained from large parovarian cysts[3] or from *colloid cancer of the peritoneum;* a sensation of peculiar and very superficial fluctuation,[4] associated, however, here with grave symptoms of cachexia, and generally with a rapidly spreading growth. *Peritoneal abscesses* enclosed by adhesions will also, if large, give rise to several of the signs of a cancer; but the history of an antecedent local or general peritonitis, the swelling not being influenced by changes in the posture of the patient, the irregular fever, the indistinct fluctuation of the tumefaction, and its acute course, may enable us to distinguish the non-malignant from the malignant affection. In rare instances a tumor may be enormous, increase rapidly, yet be simply *fatty.* There are no means of positively distinguishing the affection.[5] *Sarcoma* cannot be told from carcinoma; it is more common in advanced age.

In some cases the malignant disease is closely simulated by *dilatation of the colon*, caused ordinarily by fecal tumors. This, though it may present but a single swelling, generally occasions several, which are commonly seated at the middle third of the abdomen, are apt to

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[1] As in the case reported by Porter, Philadelphia Medical Times, June, 1875, in which the spleen weighed twenty-one pounds.

[2] See the cases of Bright, in Clinical Memoirs on Abdominal Tumors, republished from Guy's Hospital Reports by the New Sydenham Society.

[3] Bristowe, St. Thomas's Hospital Reports, vol. xi.

[4] As in the instances recorded by Albert Robin, Bull. de la Soc. Anat., 1873, and Vidal, Bull. et Mém. Soc. Méd. des Hôpit., 1874.

[5] See St. George's Hospital Reports, vol. v., 1870, p. 253.

appear on both sides, to be movable and painless and to bear handling without pain, to change their position slightly at intervals, and to become occasionally less in size. Then, after the case has been for some time under observation, we may be able to notice large and characteristic discharges; though we must not forget that a mere sluggish state of the bowels, or even diarrhœa, may exist while the colon is dilated and perhaps filled with fecal accumulations. Sometimes the mass may be seated above the symphysis and be mistaken for a pelvic tumor. Like a cancerous growth, it may lead to complete intestinal obstruction. The tympanites and the dilatation it occasions, which may be idiopathic, produce at times fatal results.[1] The dilatation may be enormous.

Cancer of the intestine has symptoms similar both to fecal accumulation and to cancer of the peritoneum. The marked cachexia and the signs of persistent and increasing narrowing of the bowel, as shown by the flattened fæces, the blood and pus in the stools, the frequent attacks of colicky pains, and the vomiting, distinguish it from the former affection. The limitation of the swelling, the absence of dropsy, the character of the stools, the frequent change in the position of the tumor and in its distinctness,[2] and, if it affect the duodenum, the decided jaundice, separate it from peritoneal cancer.

SECTION V.

ABDOMINAL PULSATION.

Aortic Pulsation.—By far the most frequent cause of a pulsation visible in the abdomen, and especially at the epigastric region, is a throbbing of the abdominal aorta. It is common in neurasthenics and hysterical persons. Some women are liable to it immediately before their menstrual periods or during the earlier months of pregnancy. In men it is seen most often in those who suffer from inveterate dyspepsia, and is apt to come on in severe paroxysms, which are alarming to the patient, but which generally disappear under brisk purging. In hypochondriacs whose abdominal walls are thin, the beating at the epigastrium may become a source of continued distress. The increased action of the aorta, or, as happens in emaciated persons, the greater distinctness with which the beat of the artery is perceived

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[1] Gee, St. Barthol. Hosp. Rep., vol. xx.; A. Money and S. Paget, Clin. Soc. Transact., 1888; Formad, Trans. Coll. Physicians, Phila., 1892.

[2] Leube, Ziemssen's Cyclopædia.

without there being abnormal throbbing, may be distinguished from an enlarged and somewhat displaced heart by the circumstances of the case and the absence of the physical signs of cardiac disease; and from an aneurism by the want of the signs that characterize an aneurism.

Abdominal Aneurism.—Aneurism of the abdominal aorta is a disease of middle life, and of males especially. Its most frequent cause is excessive muscular exercise; sometimes it is produced by a blow on the abdomen, or by syphilis. Its duration is very uncertain; occasionally six or seven years elapse from its earliest indications until the fatal termination; not unusually the patient lives twenty to thirty months after its occurrence.

The chief *symptoms* are pain, and an absence of dropsy, of fever, or of any considerable constitutional disturbance. The pain is generally felt in the back, or in the right hypochondrium, or shooting down the sciatic nerves to the lower limbs. It may be constant and dull, or occur in protracted and violent paroxysms; ordinarily there is a persistent pain which has periods of fierce exacerbation. The disproportion between its violence and the otherwise almost unimpaired health is a striking feature of the disease, and continues until the aneurism becomes very large and occasions displacement of important organs. Besides pain, vomiting and hiccough are sometimes prominent symptoms.

The *physical signs* of an abdominal aneurism are: an impulse communicated to the hand when placed over the swelling; a systolic blowing sound; a thrill; and in some instances a distinct prominence and alteration in the form of the abdomen. The impulse corresponds, with rare exceptions, to the beat of the heart, is single, and ordinarily very forcible. Generally it cannot be felt from behind; it is a beat discerned only anteriorly and on either side of the pulsating sac. With the expansion of the tumor, we hear a short blowing sound, both posteriorly and anteriorly, sometimes perceived in the recumbent posture only; or a dull, muffled sound; rarely are there two sounds. A thrill felt at the same time as the pulsation is noticed; still, it may be absent, even in large-sized aneurisms. The pulse in the femoral is often retarded.

Aneurism of the abdominal aorta may be confounded with—

RHEUMATISM; NEURALGIA; COLIC;
DISEASE OF THE SPINE;
AORTIC PULSATION;
LUMBAR AND PSOAS ABSCESS;
NON-ANEURISMAL PULSATING TUMOR.

The first four of these affections are likely to be mistaken for an abdominal aneurism, on account merely of the pain; the others, because of the presence of pulsation, or of a swelling, or of both pulsation and swelling.

Rheumatism; Neuralgia; Colic.—The pain caused by an aneurism may closely simulate rheumatism of the lumbar muscles, or sciatica, or abdominal neuralgia, or colic. There is nothing in the pain itself which will lead to the detection of its origin: this can be effected only by a recognition of the physical signs of the aneurism. Yet, abdominal pain, or abdominal neuralgia, especially when obstinate, must always make us very suspicious of an aneurism. In doubtful cases a skiagraph may prove of much value.

Disease of the Spine.—Patients who are suffering from aneurism often complain of pain in the spine, and present sometimes an obvious spinal curvature. But a careful examination, by detecting the physical signs of an aneurism, will enable us generally to distinguish the source of the difficulty. The constant boring pain so much complained of in cases of aneurism is usually thought to be due to absorption of the vertebræ, but it has no necessary connection with this lesion.

Aortic Pulsation.—Simple abdominal pulsation, such as we observe in neurasthenia, hysteria, in dyspepsia, in pregnancy, and in movable kidney; or excessive epigastric pulsation due to an enlarged right ventricle or to insufficient aortic valves, may be readily mistaken for an aneurism. But in the former case the history will generally lead us to a correct conclusion, especially if taken in connection with the facts that the pulsation is not heavy and slow, as in an aneurism, but jerking and sudden; that there is no thrill; no tumor with corresponding dulness on percussion, if we except pregnancy; no systolic murmur audible in front of the abdomen or along the spine; and no pain.

The pulsation due to disease of the heart is discriminated by the physical signs in the thorax. Regurgitation at the aortic orifice, which is the cardiac affection most liable to be confounded with an aneurism, on account of the marked pulsation it may occasion in the left hypochondrium or at the anticardium, is distinguished by the single or double blowing sounds, which are heard not only over the thorax, but also over many arteries of the body, and by the character of the pulse.

Lumbar and Psoas Abscess.—In some cases, soft, fluctuating, deep-seated tumors, that are really produced by an aneurism, may arise in the lumbar region; nay, they may seem to point, like a psoas abscess, at Poupart's ligament. But, unlike an abscess, the effusions of blood give rise, with rare exceptions, to impulse and to murmur.

Non-Aneurismal Pulsating Tumors.—When a tumor of any kind presses upon the aorta, a distinct pulsation is communicated, and the similarity to an aneurism is heightened by the circumstance that the morbid growth may produce a murmur. The tumors which most usually occasion the phenomena mentioned are: enlargement of the left lobe of the liver, cancer of the pylorus, disease of the pancreas, or of the omentum, or of the mesentery, and, in rarer instances, enlargement of the kidney, fecal accumulations, and cancer of the lumbar glands. To avoid error, we must pay close attention to the history of the disorder and the attending gastric and renal symptoms; we must trace, by percussion, the outline of the solid mass, and see if it correspond with any viscus. Then, in non-aneurismal tumor the patient has almost always been in bad health before the tumor is detected, and the swelling rarely causes pain of such severity as is observed in an aneurism; moreover, the transmitted aortic impulse is lessened by placing the patient on his hands and knees, thus taking away the pressure from the artery. A varicose state of the epigastric veins and the existence of ascites will also decide against an aneurism; while, on the other hand, the lateral as well as the forward direction of the impulse, violent neuralgic pains in the loins or shooting down the back, and an immovable tumor, are in its favor. Still, there are cases in which a morbid growth lying across the aorta occasions symptoms so nearly like those of an aneurism that the most skilful diagnostician finds himself in doubt; or cases of aneurism in which the physical signs are absent, and in which the affection affords no indication of its existence, beyond, perhaps, pain. Under these circumstances we can only suspect its occurrence.

But supposing that, from the combination of the physical signs and symptoms, we know that we are dealing with an abdominal aneurism, can we be sure that it is aortic? We cannot; for, although this is generally its seat, an aneurism of the splenic or the cœliac artery, of the superior mesenteric artery, or of the renal artery, may produce the same phenomena.[1]

When an aneurism bursts, it gives rise to symptoms which vary with the seat of the rent. The accident is always fatal, but death may not follow for several days; usually great tenderness of the abdomen and changes in the physical signs are at once produced.

[1] See Ballard, Physical Diagnosis of Diseases of the Abdomen, p. 217.

CHAPTER VII.

ON THE URINE, AND ON DISEASES OF THE URINARY ORGANS.

URINE.

THE urine, besides being the most accurate index of the condition of the urinary organs, becomes a fair indication of that of many other important secreting glands in the body. To glean the full benefit from an analysis of the urine, we must explore it not merely qualitatively, but quantitatively, and examine its deposits with the microscope. Modern chemistry is especially endeavoring to find means which will determine, by apt volumetric processes, the exact proportion of the ingredients as accurately and as easily as hitherto we have detected their presence. This is a subject which cannot be more than indicated in these pages: only such of these investigations will be noticed as have furnished results which may be made readily available for the exigencies of professional life.

It is customary, in quantitative analyses, to use the French system of measures, and to employ instruments on which cubic centimetres are marked. One thousand cubic centimetres are equal to one litre. or 2.1 pints, or to a thousand grammes of water; and one gramme is equal to 15.434 grains; one centigramme to .1543 of a grain.

Urine, in its normal state, is an amber-yellow fluid, of acid reaction, and specific gravity of 1016 to 1020 as compared with distilled water at 1000. On standing from eight to twelve hours, a slight cloudy deposit takes place, consisting mainly of mucus, epithelial cells from the urinary passages, and a few crystals. Normal urine freshly voided contains no bacteria, and is aseptic.

Ordinarily, urine soon undergoes decomposition, which renders the results of analysis valueless. It is advisable, therefore, to examine every specimen promptly, but, as this cannot always be done, the addition of some preservative may be needed. Chloroform seems to be the most suitable; six or eight drops added to each fluidounce, the mixture to be well shaken, will preserve samples for months, even in hot weather. Chloroform gives a strong reaction similar to sugar with Trommer's test, but does not reduce bismuth subnitrate nor interfere

with the phenylhydrazin test. It arrests the fermentation of sugar and of urea.

In the examination of sediments great advantage, both as to time and complete collection of the suspended matters, is gained by the use of a centrifugal machine, several forms of which are now procurable. The electric centrifuge is the most convenient. The centrifugal method tends to exaggerate the amount of material, as compared with the old method of sedimentation, but by it we may obtain casts and suspended matter which otherwise would be missed. In addition to its usefulness in urine-examination, a good high-speed centrifugal machine is of much use in other clinical work, especially in examining sputum and blood. Purdy's percentage tubes increase the advantage of the instrument.

The manner of obtaining a specimen of urine is not unimportant. We should instruct our patient, as is so strongly recommended by Sir Henry Thompson,[1] to pass the first two ounces into one vessel, and the remainder into another. We thus procure a specimen of the renal secretion, in addition to anything in the bladder, separate from any urethral products, and avoid the error of confounding prostatic or urethral with vesical or renal disease. When it is essential to obtain a specimen of urine absolutely pure and unmixed with products of the bladder, the same authority recommends the drawing off of the urine by means of a soft gum catheter, while the patient is standing. The bladder should then be carefully washed out by repeated one-ounce injections of warm water. The urine is now to be permitted to pass, as it will do, drop by drop, into a small glass vessel. The bladder contracts around the catheter, and the urine percolates direct from the ureters, through their virtual prolongation,—the catheter,—into the receptacle. The urine passed in the morning, immediately after rising, will be found to represent with sufficient accuracy the general process of disassimilation; but, if greater accuracy be desirable, a specimen of the mixed urine of the twenty-four hours should be used.

As regards the *quantity* of urine daily voided, the mean average of healthy persons is 1500 cubic centimetres (fifty fluidounces). In summer, when the skin is acting freely, less fluid passes off by the kidneys than in winter. The more liquid that is taken into the system, the greater is the secretion of urine, unless the other organs that eliminate water, as the skin, the lungs, and the intestines, are excreting with unwonted activity.

The quantity is diminished in all cases in which the specific gravity is increased, with the exception of diabetes; it is diminished in acute

[1] Clinical Lectures on Diseases of the Urinary Organs.

diseases, in fevers, in cholera, and in the early stages of dropsies; in some forms of Bright's disease, particularly the acute forms, through their entire course, and often in the last stage of all forms of that disease. It is, on the other hand, augmented in cardiac hypertrophy and whenever the specific gravity is diminished; in hysteria; in contracted kidney, and in polyuria. In almost all vesical and renal affections frequent micturition is a marked symptom,—not always, however, associated with increased quantity of urine.

The *ingredients* of urine are numerous. The principal are: urea, sulphates, phosphates, chlorides, uric acid and urates, kreatinin, hippuric acid, mucus, coloring-matter, and a large proportion of water.

The following data for average normal urine are taken from an article by Charles Platt;[1] the ingredients are given according to a strictly scientific system.

Reaction, acidity in twenty-four hours equivalent to 2–4 grammes of oxalic acid. Total quantity of liquid in twenty-four hours: man, 1450 cc.; woman, 1250 cc.

	Grammes excreted in twenty-four hours.	
	Man.	Woman.
Total solids	60.0	51.0
Urea	34.0	30.0
Uric acid	0.6	0.5
Kreatinin	0.9	0.8
Hippuric acid	0.7	0.6
Xanthin and analogues	0.005	
Minor organic matters including pigment	0.3	
Sulphur dioxide derivable from ethereal sulphates	0.250	
Chlorin	7.3	6.0
Phosphoric anhydride	3.0	2.5
Sulphuric anhydride	2.2	1.9
Potassium oxide	3.0	2.8
Sodium oxide	4.5	4.0
Calcium oxide	0.3	0.28
Magnesium oxide	0.4	0.35
Ammonia (NH_3)	0.7	0.6
Iron	0.007	

Besides the elements mentioned, the quantities of which fluctuate with the food-supply and with the activity of tissue-metamorphosis, we meet, in morbid states, with substances that do not exist at all in healthy urine, or the presence of which is doubtful, such as various forms of albumin, sugar, blood, bile, fats, oxalate of lime, and certain pigments. Most of these are dissolved in the urine, and are not to be

[1] Journ. Amer. Chem. Soc., 1897, p. 382.

detected except by delicate tests; others form in sediments after the urine has been discharged, and may be recognized by the microscope.

As matters of clinical interest we endeavor to fix these waymarks: the color, the specific gravity, the quantity, the reaction, the presence or absence of such important abnormal ingredients as albumin and sugar, and the character of the deposits. Frequently, too, we extend our examination until we have determined approximately, if not accurately, the increase or diminution of the main constituents of the urine, especially of the urea, uric acids, chlorides, phosphates, and sulphates, and the distribution or non-distribution of bile and other unusual constituents through the fluid.

Color.—The color of the urine is much affected by food and medicine, as well as by various morbid processes. A smoky or a red aspect is apt to be owing to admixture of blood; a very light color denotes generally an increase of water, and is commonly found in diabetes, in hysteria, and in kindred nervous affections. In febrile diseases the urine is of dark hue. A greenish-yellow or brownish tint of the discharge is indicative of bile; but a similar tinge may be present when rhubarb has been taken. A dirty-blue urine happens from an indigo sediment, and is alkaline. Strong coffee darkens the urine; turpentine darkens and imparts a violet color to it; carbolic acid, tar, and creosote render it black; so do disintegrated blood and melanotic cancer. Santonin, logwood, and senna discolor it. The first-named substance gives it a bright yellow color, which on the addition of an alkali becomes crimson. Senna may impart to it a brownish or a deep red color, which, however, like that due to rhubarb, is lightened on the addition of mineral acids, and is thus distinguished from the hue of urine containing blood. The altered appearance is mostly due to the coloring-matter of these articles being excreted with urine.

The chemistry of the coloring-matters of the urine is still incomplete, and the clinical significance of the color-changes still obscure. The principal normal coloring-matter is *urobilin*, which is an oxidation-product from blood and bile-pigment. In febrile conditions a less oxidized product is excreted, which MacMunn has named *pathological urobilin* and declares to be identical with the coloring-matter of the fæces, stercobilin. He further states that the presence of this body in the urine is to a certain extent an indication of the absorption of fecal matter and ptomaines which have not been destroyed by the liver. Other pigments have been described, among which may be named *uroerythrin*, *urochrome*, and hæmatin free from iron, *hæmatoporphyrin*, and *melanin*, which occurs especially in the urine in melanotic cancer and wasting diseases. The employment of the

spectroscope is one of the means of distinguishing between these colors, but a description of their minute differences would be beyond the scope of this work.

Specific Gravity.—We take the specific gravity of urine to judge of the solid matter it contains. The readiest means is the urinometer. For the implement to yield trustworthy results the fluid should be brought to the temperature at which the urinometer has been graduated. A difference of seven degrees F. corresponds to about one degree of the urinometer. Most instruments are graduated for use at 60° F.; the cheaper forms are often inaccurate. Squibb makes a urinometer adapted for use at 77° F. (25° C.), which is convenient for office work. More accurate than the urinometer is the specific gravity bottle, or the Westphal balance.

If there be but a small quantity of urine for examination, we note the amount and how many volumes of distilled water it takes to fill the vessel to the height required to float the urinometer. We then multiply the number above 1000 that the instrument shows, by the total number of volumes of the mixed fluid. This is only approximate.

From the specific gravity we may calculate approximately the quantity of solid matter passed by multiplying the number above 1000 by 2.33. This may be done whether we estimate in grammes or in grains. For instance, in urine of specific gravity of 1010 there will be 23.3 grammes of solid matter in each 1000 grammes of urine; in urine of 1030, 69.9 grammes. This information obtained, it is easy to find the whole amount of solids contained in the urine of twenty-four hours, after ascertaining the quantity passed in that time. To take the first illustration: if 1000 grains yield 23.3 grains of solid matter, how much would be yielded by 20,000 (the quantity passed, let us say, in twenty-four hours)?

$$1000 : 23.3 : : 20{,}000 : x. \quad x = 466 \text{ grains.}$$

This method is not very precise; when exactness is required, the urine must be evaporated until a dry residue is left, which should then be carefully weighed.

The amount of solids in healthy urine is variously estimated. The table above given exhibits a fair average. As a rule, the proportion is greatest in persons of heavy weight; if, therefore, we wish to make nice comparisons, the weight of the body should be always stated. To ascertain how much of the solid matter consists of mineral matters, the organic substances must be burned off at a red heat.

In disease, the solids, and with them of course the specific gravity,

fluctuate very much. We find the specific gravity decidedly increased, rising to 1030 or higher, when sugar or an excess of urea is present, and when the urine is concentrated and of deep color. A low specific gravity is met with in chronic interstitial nephritis, in many cases of hysteria, and in pale urine except that of diabetes. But to be accurate—and, indeed, accuracy in regard to the other physical and chemical properties is unattainable without attending to the same rule—we must not lay stress on the specific gravity without taking into account the measure of urine passed in the twenty-four hours.

Reaction.—Normal urine is acid. The *acidity* depends upon acid salts, especially acid sodium phosphate. The degree of acidity is not always equal, and is much influenced by digestion. If no food have been taken for hours, the discharge is highly acid; that passed after a meal, and while the process of digestion is going on, is but faintly so, or even alkaline. In about three or four hours after meals the alkaline tide turns, and the acidity of the urine slowly increases until food is again taken. There seems, however, to be a limit to the increase, for Bence Jones found that continuing to fast for twelve hours beyond the usual meal-time did not intensify the acidity of the urine. The alkalinity of the urine after meals is rarely detected at the bedside. For, although the urine may be alkaline when secreted, it is generally mixed in the bladder with that which collected before or after the alkaline tide, and the mixed urine when passed may have an acid reaction. The acidity of the urine is augmented by the administration of the vegetable or the mineral acids; yet they do not cause, even in large doses, as great variations as does digestion. We find the urine very acid during a meat diet; the acidity is also strongly marked if any acid be present in the urine which sets the uric acid free, or if this be in decided excess.

For determining reaction, litmus-paper is used. Solution of litmus is divided into two parts; to one part nitric acid is added, drop by drop, until the color is wine-red. This is then mixed with the other half. Slips of filtering-paper are dipped in this and dried. They have a purple tint, and are very delicate, responding to a trace either of free acid or of alkali. We thus avoid the use of two colors. Where litmus-paper of two colors is used, we find that the blue is turned red by an acid; the red, turned blue by an alkali. Litmus-paper is best kept in a closed dark bottle.

We may estimate the amount of free acid in the urine by a solution of sodium hydroxide (caustic soda) containing 4.0 grammes to the litre. This solution is added drop by drop to 100 cc. of urine, which has been measured off in a beaker glass. After the addition of each

half cubic centimetre, a drop of the mixture is placed, by means of a glass rod, on well-prepared litmus-paper. When the paper is no longer reddened, the analysis is finished; and by noting how much of the standard solution has been used, we can determine the acidity of the urine, which it is customary to express as equal to so many grains of oxalic acid, the value of the sodium hydroxide solution in terms of oxalic acid having been previously ascertained.

Urine, when voided, remains ordinarily acid for at least a day; but it may lose its acidity much sooner. This is always a significant fact, having much the same meaning as if the fluid had been discharged in a neutral or an alkaline state.

Now, an *alkaline* reaction may result from several causes: from the effect of digestion, as already mentioned; from the presence of sodium or potassium carbonate; or from the decomposition of the urea into ammonium carbonate. In the former case, heat does not restore the color of the red litmus-paper,—it remains blue; in the latter, a gentle heat soon brings back the original red tint. Moreover, in either case, the earthy phosphates are precipitated, the fixed carbonate causing the precipitation of the amorphous calcium phosphate; while, by the ammonium carbonate, ammonium and magnesium phosphates, in conjunction with the calcium phosphate, are thrown down, and the triple phosphate is abundantly formed, and can be easily recognized under the microscope by its prismatic crystals.

Alkalinity of the urine from fixed alkali is not inconsistent with health. We have adverted to the effects of digestion and to the fact that alkaline urine results from the use of certain articles of vegetable food, or of the salts of sodium and potassium. Urine owing its alkalinity to ammonium carbonate is always to be viewed as pathological. The disturbance is generally long continued, and the urine loses its acidity in the bladder, in consequence of a disease of the mucous coat of the viscus, or from being long retained there, as in cases of paraplegia, or from admixture with pus, which acts as a kind of ferment and leads to decomposition of the urea.

Changes in the Quantity of the more Important Constituents of Urine.—*Urea.*—The amount of urea excreted by well-nourished, healthy, adult males in the twenty-four hours is estimated, in round numbers, by Roberts at 3½ grains per pound weight of the body, and by Neubauer and Vogel at 25 to 40 grammes, or 0.37 to 0.6 gramme for every kilogramme of weight of the body. Purdy places the mean excretion of urea in healthy adult males between the ages of twenty and forty years at 33.18 grammes (512.1 grains) in twenty-four hours. These figures are like those given by Platt.

Urea is the principal product of the transformation of nitrogenized substances. Its proportion fluctuates, therefore, with the variations in the nature and quality of food partaken of, as well as with the activity of the transformation of the structures of the system: hence it becomes the most important index of the waste and repair of tissues. Exertion of body and of mind leads to the discharge of a larger quantity of urea. If this be replaced by a nourishing diet, nothing is lost; the body retains its health. But when the requisite amount of nitrogenized aliment is not taken, or, if taken, cannot be assimilated, owing to a disturbance in digestion, the person wastes. We notice, too, in acute febrile states, until their height is reached, hand in hand with the emaciation an increase of this significant urinary constituent,—a proof, then, of the rapid and unsupplied disintegration of the tissues. We see the same increase during paroxysms of intermittent fever, in inflammations, and in some cases of nervousness; also from a predominant animal diet, and in certain forms of indigestion, in which the food is speedily passed off in the shape of urea instead of acting its part in the nutrition of the economy. Degenerative changes in the liver may be accompanied by a diminution of urea-excretion.

A lessened quantity of urea is excreted during fasting, while on a vegetable diet, in dropsies, and in many long-continued organic diseases that gradually undermine the general nutrition and diminish tissue-change, or in states attended with diminished oxidation. But the decreased amount in the urine may also be due to a want of secreting power of the kidneys. The urea, or the products of its decomposition, then act as a poison in the blood; and the symptoms indicative of uræmic poisoning are encountered. Urea is sometimes not found in the urine at all, or only in traces, having been replaced by leucine and tyrosine.

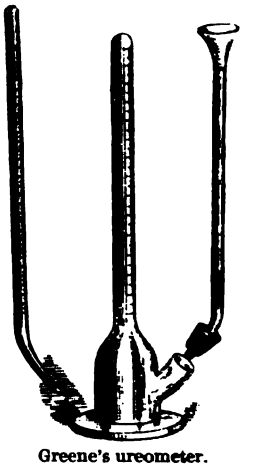
Quantitative estimations of urea are almost exclusively made by the use of solutions of sodium hypochlorite or hypobromite, which decomposes the urea, liberating nitrogen and carbon dioxide in amounts proportional to the urea present. The carbon dioxide is kept in solution by using excess of sodium hydroxide or carbonate, and the volume of nitrogen is measured. The most accurate results seem to be obtained with the hypobromite, but this does not keep well, and its extemporaneous preparation is troublesome and annoying. Sodium hypochlorite is readily obtained, being the common Labarraque's solution. It keeps in good condition for a long while, and gives good results. It must contain a marked excess of sodium carbonate. Several observers have reported that improved effect is obtained from the addition of potassium bromide (1 gramme to 25 cc.

of *liquor sodæ chloratæ*). To avoid the annoyance of using pure bromine, when hypobromite solution is employed, Charles Rice suggested the use of a solution of bromine in potassium bromide. This keeps well, and is more convenient to handle. In this method the solutions used are as follows: (*a*) 10 grammes of potassium bromide are dissolved in 80 cc. of water, 10 grammes of bromine added, and the liquid shaken until the latter is dissolved; (*b*) 10 grammes of sodium hydroxide are dissolved in 25 cc. of water. For use, equal quantities of the two liquids are mixed and slightly diluted with water. Sodium hypobromite may also be prepared by adding directly 1 cc. of bromine to 25 cc. of the above solution of sodium hydroxide; but the liquid

FIG. 56.

Doremus's ureometer.

FIG. 57.



Greene's ureometer.

must be used within a few hours, or accurate results cannot be expected. The mixture must be made in a well-ventilated place, as bromine is exceedingly irritating and corrosive.

For collecting and measuring the nitrogen evolved, many kinds of apparatus have been devised. That of Hüffner is a standard model, but simpler and less expensive forms are now usually employed in clinical work. Fig. 56 shows a form devised by Doremus, which is much used. The apparatus is filled with solution of hypobromite or hypochlorite, so that when the graduated tube is upright the bulb is about half filled. A large watch-glass or shallow dish should be placed beneath to catch any overflow. A measured quantity of the urine (1 cc.) is introduced by means of the dropping-tube, the opening being pushed well into the bend of the upright tube, and the apparatus

being tilted forward to prevent any escape of bubbles or urine into the large bulb. After about twenty minutes the volume of gas is read off; 1 cc. of nitrogen may be taken to represent .0028 of urea, but the tube is usually graduated so as to read directly the percentage of urea, a definite volume of the urine being taken for each test. An improved form of Doremus's ureometer is now obtainable. Fig. 57 shows Greene's ureometer, also a simple instrument.

Fowler's method has been endorsed by several careful observers. It depends on the fact that the decomposition of urea greatly reduces the specific gravity of the urine. It may be performed as follows. The specific gravity of the sample is carefully taken, and then 25 cc. added in a large beaker to 175 cc. of solution of chlorated soda (U.S.P. 1890), and, after mixing well, allowed to stand for a few hours, when the specific gravity is again taken. Multiply the specific gravity of this residual liquid by 7, add the specific gravity of the original liquid, and divide the sum by 8, subtract from this quotient the specific gravity of the residual mixture, multiply the remainder by 0.77, and the product is the percentage of urea. In case the urine is of high gravity it is better to use 12.5 cc. diluted with an equal volume of water and then add the 175 cc. of solution of chlorated soda. The result must be multiplied by 2.

Uric Acid.—Uric acid, like urea, is a product of the metamorphosis of tissue. It was supposed by Liebig that the acid is an early stage of the transformation of urea. Hofmann teaches that uric acid is deposited owing to the decomposition of the urates by the acid phosphate of sodium. Under ordinary circumstances, the deposition of uric acid occurs subsequently to the expulsion of the urine; but should the acid sodium phosphate be in excess, the uric acid may be precipitated before the secretion is voided, and thus give rise to gravel and calculi. This may also happen through too great concentration of the urine.

The amount of uric acid passed in twenty-four hours varies from 0.5 to 1.0 gramme. It corresponds in general to the amount of urea in the proportion of 1 to 33. In normal urine the presence of uric acid cannot be detected without the addition of a strong acid, since it exists in the form of soluble urates, which must be first decomposed. The uric acid is gradually thrown down in small red grains.

The characteristic reaction of uric acid is furnished by the *murexide test.* A few drops of nitric acid are mixed with the suspected deposit in a capsule, and the mixture is slowly evaporated to dryness, best on a water-bath; a drop of ammonium hydroxide is then added, which produces instantly a rich purple.

But both uric acid and the urates can be easily and quickly discriminated by the microscope. The crystals of uric acid are readily discerned, notwithstanding that they vary both in size and in form. Rhombic plates with rounded angles are frequent. To obtain the crystals rapidly, where they are not passed as uric acid, a portion of the suspected deposit is dissolved in a drop of potassa, and the alkaline solution treated with an excess of acetic acid; after the lapse of a few hours crystals of uric acid will be formed.

FIG. 58.

Crystals of uric acid, magnified about 200 diameters. Most of these forms are seen in the urine of acute rheumatism.

The *quantitative estimation of uric acid* is regarded by many authorities as a very important operation, and several methods have been devised for the purpose. Most of these are tedious and difficult. The following modification of more difficult methods was devised by Bartley,[1] and is satisfactory for clinical work.

The solutions required are: *Silver nitrate solution*, $\frac{n}{50}$,—that is, containing 3.4 grammes of silver nitrate in 1000 cc.

Magnesium Mixture.—Ten grammes crystallized magnesium sulphate, 12 grammes ammonium chloride, and 100 cc. *aqua* ammoniæ, U.S.P.

Ammonium Hydrosulphide or Potassium Sulphide.—This solution should be freshly made, and of such strength that its color is that of the urine.

The analytic process is as follows: When the sample shows a sediment of uric acid or urates, it should be warmed with a few drops of sodium hydroxide to dissolve these, the liquid stirred, and

[1] Medical Chemistry, 5th edit., p. 641.

the excess of alkali neutralized by acetic acid. In operating on very dark urines it is well to dilute with an equal volume of water. The titration is performed in hot solution to avoid precipitation of the xanthin bases.

To 50 cc. of the clear urine add 5 cc. of the magnesium mixture and about 10 cc. of ammonium hydroxide (U.S.P.),—that is, enough to give a decided excess. Heat the solution on a water-bath and add the silver nitrate solution in small amounts from a burette. Between each addition remove a drop of the warm liquid by means of a dropper pipette, over the end of which a bit of absorbent cotton has been tightly wound to serve as a filter, and, after removing this filter, bring the drop in contact with a drop of the sulphide solution lying on a white plate. This testing is continued until the removed drop gives a dark ring or cloud on contact with the sulphide solution. The number of cubic centimetres of silver solution is then read off and 0.5 cc. deducted to allow for the amount of solution required for a perceptible reaction in the absence of uric acid. Each cubic centimetre of silver solution corresponds to 0.00336 grain of uric acid.

As soon as the process is complete, the precipitate settles rapidly, and it is well to draw off some of the clear liquid and test again; or a drop of the silver solution may be added to make sure that no further precipitation will occur. When the solution cools, however, additional silver solution must be added before the end-reaction is obtained, since the xanthin bases then react. By making two titrations, one with the hot liquid and one with the cold, the excess of cubic centimetres in favor of the latter, being multiplied by 0.0015, will give the amount of xanthin bases.

In disease, the fluctuations in the quantity of uric acid are great; as a general rule, they correspond to the rise and fall of urea. We find the acid diminished in hydruria and in affections in which the eliminating power of the kidneys is interfered with, as in the more advanced stages of Bright's disease and in anæmia and chlorosis. An increase is encountered in acute inflammations, in fevers, in functional disorders and many of the structural affections of the liver, in heart and lung diseases attended with dyspnœa, in leukæmia, and in acute rheumatism.

We must, however, be careful not to suppose the uric acid to be in excess because it is readily precipitated. It may or may not be in larger amount: the sediment merely proves an augmentation of acidity in the urine sufficient to take away the base from the uric acid. This happens often as the result of acid fermentation of the urine. Frequently urates are separated along with the uric acid; we find

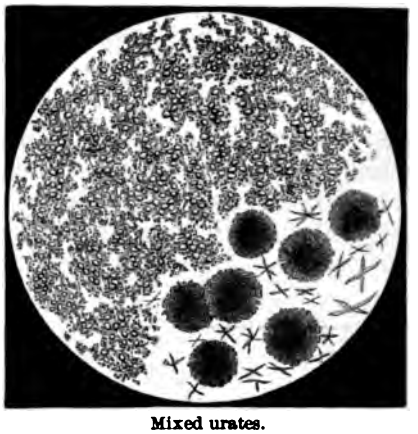
then generally a dark urine of high specific gravity and of very acid reaction.

Persons who habitually pass urine of the character described are subject to gastric or hepatic disorders. They are also often gouty, or lithæmic, and frequently consumers of a large amount of animal food, or intemperate or indolent in their habits.

Uric acid or urates are never found as sediments in freshly voided healthy urine. Occasionally precipitates of uric acid or urates occur in the urinary passages. Now, these sediments may concrete and form the nuclei of calculi; or they may be passed in small particles commonly spoken of as "gravel."

Urates.—The pathological conditions in which the urates are changed are much the same as those in which alterations in uric acid

FIG. 59.



Mixed urates.

occur. The urates are principally the sodium, potassium, and ammonium urates. The deposits formed by their precipitation are of pink color, sometimes brown, or like brick-dust, or yellowish, or even white. From pale urine of low specific gravity a white sediment is apt to settle. All the deposits are dissolved with readiness by heat. Acids decompose them and separate uric acid. They are all more soluble in warm water than in cold, and the neutral salts are more soluble than the acid ones.

Under the microscope, the urates are seen to be either irregular, amorphous particles, needle-like crystals, dumb-bells, or round globules of various sizes, from some of which fine needles project. The latter, like the dumb-bells, are commonly supposed to be sodium urate; the globules and crystals, sodium urate and ammonium urate;

the granular, amorphous powder, mixed urates, more especially sodium urate and potassium urate. These amorphous urates may, under the microscope, be mistaken for calcium phosphate. The differential test consists in their behavior with acids: the phosphate is dissolved by acetic or hydrochloric acid; the urates are gradually transformed into crystals of uric acid. Then, a deposit of calcium phosphate is often more cloudy than the urates, and, unlike them or uric acid, is not soluble in liquor potassæ. From calcium carbonate, which also occurs in a granular form, both the urates and the calcium phosphate are distinguished by the effervescence of the carbonic acid which happens on the addition of a strong acid.

Urine containing a sediment of urates is generally markedly acid, or soon becomes so, either from an absolute increase of the uric acid, or in consequence of changes in some of the constituents of the fluid —as of the pigment—which take place either before or shortly after emission. Not infrequently, too, it is scanty, and the urates are deposited as soon as the urine cools to the temperature of the atmosphere. Their precipitation may be, and indeed often is, due to there not being water enough to hold them in solution. We may judge of this being the case by ascertaining the amount of urine passed in twenty-four hours. If the quantity be about normal, the deposit is in all likelihood due to an excess of urates. In cold weather these deposits occur more quickly and more extensively than in warm.

Sediments of urates are at times met with in pale urine, and without either diminution of water or excess of acidity. The urine yields but a faintly acid or a neutral or an alkaline reaction, and under the latter circumstances calcium phosphate, or even triple phosphates, may be observed to accompany the urates. The urate present is acid ammonium urate.

Phosphates.—The phosphates are derived in part from the food, in part from the disintegration, or rather the oxidation, of the disintegrated albuminous substances, and especially of the nerve-structures. They occur either as calcium and magnesium phosphates, the *earthy phosphates*, which exist in small amounts, about one gramme in twenty-four hours, and as sodium phosphate, about three times as abundant, forming the greater part of the *alkaline phosphates.*

In health the phosphates are kept in solution by their acidity; but as soon as the urine ceases to be acid they are deposited. Hence the appearance of phosphates bespeaks a neutral or an alkaline condition of the urine, with the exception that calcium phosphate may occur in acid urine. Often the fluid, as we have already seen, becomes alkaline from the decomposition of the urea into ammonium carbonate. This

acts upon the phosphate, forming ammonio-magnesium phosphates, which crystallize commonly in transparent prisms or in feathery-looking bodies, easily distinguished from the amorphous powder or small round globules of calcium phosphate. Yet there is, as Roberts has pointed out, a crystalline form of calcium phosphate which might be mistaken for one of the stellar forms of uric acid, but it may be distinguished by its being invariably colorless. These earthy phosphates are all readily soluble in acids, even in weak acids like acetic acid, and this at once distinguishes them, even under the microscope, from calcium oxalate, which some forms resemble. In many specimens of urine they are precipitated by heat; but the addition of an acid soon dissolves them, and thus prevents the turbidity from being mistaken for that due to albumin.

FIG. 60.

Earthy phosphates; the granules are chiefly calcium phosphate, the rest triple phosphates.

The triple phosphates are often met with in heavy deposits mixed with pus, especially in the alkaline purulent urine resulting from chronic vesical catarrh. They are also seen in cases of retention of urine due to temporary or permanent paralysis of the bladder, as in low fevers, in hemiplegia, or in paraplegia. They are found, too, in many affections in which the vital powers have been seriously lowered and the acidity of the urine diminished, as during convalescence from acute disease. Under the latter circumstances, and in fact whenever the urine has become alkaline from the presence of a fixed alkali, the phosphatic deposit shows a large excess of the amorphous phosphates, if, indeed, it do not altogether consist of them.

Urine alkaline from fixed alkali, and depositing phosphates, is, unless this condition have been brought about temporarily by fruit or other food, a matter of serious import. We encounter it in persons

laboring under great general debility and indigestion associated with an impaired tone of the nervous system, and with aching pains in the lumbar region and a tendency to boils,—in fact, in those of whom it is customary to speak as exhibiting the phosphatic diathesis, or as having *phosphaturia*. Such a morbid state is not uncommon in men depressed by mental toil or anxiety, and may become associated with harsh, dry skin, thirst, enormous flow of urine and marked emaciation, giving rise to so-called *phosphatic diabetes*. The excretion of phosphates may be from seven to nine grammes daily; the urine is usually acid. In some cases there is also sugar, or this makes its appearance subsequently.

In spite of the distinct sediment of the phosphates, it is sometimes doubtful if the latter are really increased in quantity. The want of the acidity of the urine permits their precipitation, and causes them to become readily apparent. On the other hand, the phosphates may be actually in excess, and yet this excess be concealed from view. This happens especially with the alkaline phosphates, the proportions of which change in disease much more than do the earthy phosphates, and indicate much more clearly the variations of the phosphoric acid.

Now, a real, not merely an apparent, increase of the phosphates occurs, according to Bence Jones, in acute inflammatory diseases of the nervous structure, and in fractures of the skull when an inflammatory action takes place in the brain. It also occurs after mental strain. We find the phosphates also augmented by the abundant use of animal food, and by very active exercise. The earthy phosphates are markedly increased in rickets and in extensive bone-disease; the phosphoric acid, as well as the sulphuric acid, the urea, and the sodium chloride, is excreted in less amount than in health during the course of a maniacal paroxysm, in epilepsy, and in melancholia. In gout as well as in Bright's disease, too, the excretion of phosphoric acid is diminished.

To determine the proportion of the *earthy* phosphates, a few drops of ammonia are added to the urine; soon a whitish precipitate is produced, which is not removed by heat. From the quantity of the deposit, after settling, we may form a rough estimate of that of the earthy phosphates. In an ordinary-sized test-tube a deposit one centimetre high represents a normal amount. But to ascertain the amount accurately we must employ a graduated glass, separate the precipitated phosphates by filtration, ignite them in a platinum capsule, and weigh the ash. The *alkaline* phosphates are not thrown down by alkalies, and, unlike the earthy phosphates, are very soluble in water. They are procured by taking the fluid from which the

earthy phosphates have been carefully removed by filtration, and adding to it a saturated solution of magnesium sulphate. Or we add to the urine about one-third as much of the magnesium mixture, and if the precipitate be copious, giving the fluid the appearance of cream, the alkaline phosphates are in excess; if there be merely a milky turbidity, they are normal.

From the deposit obtained in testing for the phosphates, some idea may also be formed of the quantity of *phosphoric* acid in the urine. The average quantity passed by an adult male in twenty-four hours is, according to Vogel, about 3.5 grammes, or about 54 grains. For the volumetric processes by which the amount of the acid may be determined, I refer to special treatises on the chemistry of the urine.

Chlorides.—The chlorides in the urine are derived from the food: they correspond closely with the amount of salt ingested. In consequence, the sodium chloride—the main chloride in the urine, for it contains but little potassium chloride and calcium chloride—is, even in health, liable to great fluctuations; the mean in twenty-four hours is estimated by Vogel and Parkes at 11.5 grammes, or about 177 grains. Bischoff states the average at 14.73 grammes. Large quantities of chlorides are excreted after active bodily or mental exercise, smaller quantities when the body is at rest, as at night. In disease, very various amounts are eliminated with the urine. In cases of chronic indigestion, of dropsy, and during an ague-fit, the chlorides are diminished. In typhus fever and in acute inflammatory affections they sink to a low level, and rise again in convalescence: an increase after a diminution is thus always a favorable sign. We may study these changes in pleurisy and pericarditis, but especially in pneumonia. At the period of hepatization the chlorides are absent from the urine, and appear in increased quantity in the sputum; during resolution they reappear in the urine.

Sodium chloride is detected by acidulating the urine with nitric acid and adding a solution of silver nitrate; a dense white precipitate of silver chloride quickly appears, insoluble in nitric acid, but soluble in ammonia. The amount of the chloride is approximately estimated by comparison with healthy urine, or by employing the method of Hofmann and Ultzmann. According to this, if in using a solution of silver nitrate of definite strength, 1 to 8, we find curd-like masses of silver chloride falling to the bottom, which on shaking the glass do not separate, we judge the chlorides to be in normal amount. If the precipitate of silver chloride be small, $\frac{1}{10}$ per cent. or less, a simple milky turbidity arises and no curdy mass deposits; whereas if the chlorides be entirely wanting there is neither milky cloud nor tur-

bidity. If the urine contain much albumin, this should be coagulated and removed by filtering before the test is applied.

Sulphates.—Sulphates are found in the urine in large quantities. They consist of potassium sulphate and sodium sulphate, the former in excess. Like the alkaline phosphates, they are soluble in the urine. To detect them, a few drops of nitric acid are added to urine, and subsequently from fifteen to twenty drops of a saturated solution of barium chloride, when a white precipitate insoluble in acids occurs. If there be merely an opaque milky cloudiness, the sulphates are in normal quantity.

The sulphates are obtained in part from the food, in part from the oxidation of the sulphur entering into the constitution of the albuminous substances of the body and the subsequent union with a base of the sulphuric acid which is formed. They are enhanced by an exclusively animal diet, after violent exercise, in acute rheumatism, in pneumonia, and in all acute febrile processes with large excretion of urea; in fact, their increase is apt to go hand in hand with that of urea. The administration of potassium raises in a striking degree the proportion of the sulphates. The sulphates show decrease during an exclusively vegetable diet and in urine of low specific gravity.

The average daily quantity of sulphuric acid passed in the urine is about two grammes. Vogel gives an easy method of determining approximately whether it is increased or diminished. After ascertaining the whole amount of urine in twenty-four hours,—say it is 2000 cc., and then each 100 cc. would contain 0.10 gramme of sulphuric acid, —100 cc. are rendered acid, and as much of a test-solution of barium chloride[1] is added as corresponds with 0.05 gramme of the acid. The mixture is now filtered, and if the filtered liquid be not made turbid by the barium chloride, we may infer that the patient has secreted less than one gramme of sulphuric acid in the twenty-four hours. If the liquid, however, be rendered turbid by barium chloride, a further quantity of this agent, corresponding with 0.5 gramme of sulphuric acid, is added; and if the filtrate be still rendered turbid, it is evident that the quantity of sulphuric acid is greater than normal. In addition to the sulphates proper, the urine contains small quantities of derivatives of sulphuric acid, known as the ethereal sulphates, one of which is phenylsulphuric acid. The origin of these bodies is believed to be in some way connected with the action of putrefactive processes

[1] Made generally by dissolving 30.5 grammes of crystallized barium chloride, powdered and air-dried, and diluting the solution up to 1 litre; 1 cc. of it then equals 10 milligrammes of sulphuric anhydride.

dependent on micro-organisms, but as yet no definite information as to their exact clinical significance is at hand.

Kreatin and Kreatinin.—These substances found in the urine are purely excrementitious, and are derived from a disintegration of the muscular tissue. Kreatinin is the product of the change of kreatin. From 0.5 to 1 gramme is excreted daily.

But few observations have as yet been made on the increase of kreatin, or on its significance in showing the activity of nutrition in the muscles in health or in disease. Active muscular exercise augments the quantity; and the same effect is probably produced by all spasmodic affections, and, as Munk has shown, at the height of acute disease, while kreatin is diminished during convalescence, and in advanced degeneration of the kidneys.

Both kreatin and kreatinin are generally included, in analyses, under the head of nitrogenous bodies. Under the microscope the crystals of kreatin are colorless and transparent.

Presence of Abnormal Substances in the Urine.—Here may be mentioned the ingredients, such as bile and blood, observed in the urine in disease only; and along with them I shall notice those constituents the occurrence of which in healthy urine is occasional, but of which it is certain that their presence in any marked degree is abnormal.

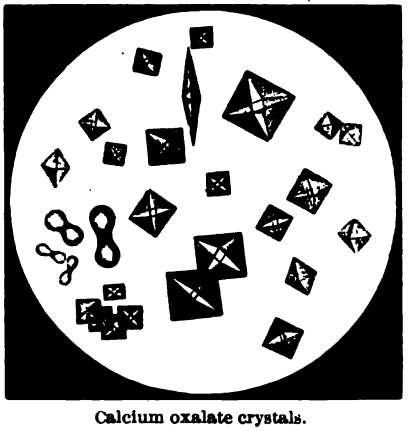
Oxalate of Lime, Calcium Oxalate.—There can be no doubt that the crystals are not found in large numbers except in a morbid condition. Some pass habitually a considerable quantity. They are generally persons weighed down by care and anxiety, or who overtask their brains by incessant application to study, or weaken their nervous power by excessive sexual indulgence or by masturbation. Sometimes they are troubled with frequent seminal emissions and irritation of the bladder, or they are dyspeptic, and suffer from uneasiness after meals; but the appetite may be good and the digestion unimpaired. They are always languid, and either very irritable or very dejected. Frequently they complain of loss of memory, and of a sensation of weight or of a dull pain across the loins. They are liable to boils and carbuncles, grow thin, and evidently are generally out of health. The urine is of high specific gravity, shows an increase of urea, and ordinarily a cloudy deposit consisting of mucus and the crystallized oxalates. Not infrequently traces of albumin are associated with the calcium oxalate.

This is the disorder called *oxaluria*, and is generally combined with tissue-changes and increased excretion of urea. Its existence as a separate affection has been denied; but I believe the clinical asso-

ciation of a considerable number of oxalates with the symptoms mentioned to be undoubted. The presence of uric acid and of oxalates is not uncommon in lithæmia. The origin of the oxalic acid is not certain. It is generally the product of incomplete oxidation of organic matters in the body, as well as of sugar, of starch, and of the salts of the vegetable acids. Probably in the first class of cases alone are the constitutional symptoms described present. In the others we may at times detect evidence of the irritation of a calculus, or of disease of the bladder or the kidneys. Acid fermentation of mucus in the urinary passages also occasions it.

Calcium oxalate may be detected in the urine when articles which contain it, such as sorrel and the rhubarb plant, have been eaten, or

FIG. 61.



Calcium oxalate crystals.

after the free use of tomatoes or of carbonated drinks. It may be also found in the urine of those recovering from severe acute maladies, and is encountered, but only in very small quantities, in the urine of healthy persons. But in neither instance is it permanent, nor can the presence of a few crystals be looked upon as of the least importance.

The microscope is incomparably the readiest means of detecting the salt. This appears in well-defined octahedra of varying size, and in dumb-bell bodies. The former are the more common and characteristic; for the dumb-bells are not frequent, nor is this formation peculiar to calcium oxalate. Occasionally, long or pointed octahedra or prismatic crystals are observed. All forms are unaffected by acetic acid.

The oxalates are often mixed with deposits of urates or uric acid; a fact which some use as an argument that oxalic acid is but the

direct transformation of uric acid. Sometimes—Beneke says constantly—the earthy phosphates coexist in large amount with the oxalates. Occasionally the irritation from the passage of the crystals gives rise to tube-casts. A case came under my observation years since in which a patient suffering from a protracted attack of oxaluria voided for weeks, along with the oxalates, hyaline, exudative, or small waxy casts. Neither heat nor nitric acid detected albumin. Under treatment, the crystals disappeared from the urine, and with them the casts. The urine examined ten years afterwards showed not the slightest sign of degeneration of the kidneys.

Leucine and Tyrosine.—Both these substances are the result of the decomposition of highly nitrogenous animal matter, are very similar, and are usually associated. They replace urea, and have been found in the urine only in disease, as in acute yellow atrophy of the liver, in typhoid fever, in smallpox, in phosphorus poisoning, in cancer of the liver, and in other forms of enlargement of the organ.[1] They are either spontaneously deposited, or form a deposit if a small quantity of urine be evaporated. Tyrosine is readily detected by the microscope. It crystallizes in long, very fine, shining needles, which may congregate in globular bodies.

Hofmann has proposed the following delicate chemical test for tyrosine. A solution of mercuric nitrate, nearly neutral, is to be treated with the solution suspected to contain tyrosine; if it be present, a reddish precipitate is produced, and the supernatant fluid is of a very dark rose-color. Leucine crystallizes in granular masses, consisting of roundish globules, sometimes of concentric form, and for the most part of yellowish color and resembling oil-drops, but, unlike oil, is not dissolved by ether. The chemical test for leucine is to place the suspected deposit on platinum foil and then to evaporate it with nitric acid. The residue is moistened with caustic soda, and this mixture is carefully heated over a spirit-lamp. It is gradually condensed into oily-looking drops,—a property which Scherer has pointed out as a characteristic of leucine.

Tyrosine is the parent substance from which the acid, homogentisinic acid, is formed that occasions *alcaptonuria*. In this rare disorder the urine when passed rapidly becomes of deep brown color and finally black. The fact that urine containing alcaptone reduces Fehling's solution, though only with the aid of heat, causes it to be mistaken for saccharine urine. But both the bismuth test and the fer-

[1] Vaughan and Beringer, Contributions from the Chemical Laboratory of the University of Michigan, vol i., 1882.

mentation test give negative results. Alkalies greatly intensify the brown color of the urine. The disorder does not markedly affect the general health, and frequently dates from childhood. It is most common in males.[1]

Bile.—The occurrence of bile in the urine imparts to it a very dark color. All the constituents of the bile may appear in the urine, or only the pigment, without the acids or their salts. The pigment is sometimes found transiently, and in small quantities, without yellowness of the skin: its more permanent and marked occurrence is, however, always attended with jaundice. It may be discerned before the discoloration of the skin is noticeable, and after it has lost its yellow hue. The biliary acids are not of necessity present in the urine of icterus.

The detection of the coloring-matter of bile is effected by pouring a small quantity of urine on a white plate; a drop of the yellow fuming nitric acid of commerce is then permitted to fall on the thin layer of fluid. Soon a play of color takes place, beginning with green and blue, passing to violet and red, and often finally to yellow or brown; the green is the predominant and the most characteristic of the colors. According to Frerichs,[2] this reaction may fail in cases where the other symptoms of jaundice are undoubted, owing to the bile-pigment having already passed through stages of transformation. When this is the case, the urine is at one time of a brown or brownish-red color, and becomes red on the addition of nitric acid; at another time it is of a deep red, which is converted by nitric acid into a dark bluish-red. Murchison has made a similar observation[3] in cases where jaundice has resulted from a blood-poison, and he has frequently found the urine to present these characters where there has been no jaundice, yet obvious derangement of the liver.

Heller's test is also very easily performed. In a small beaker glass containing about 6 cc. (1.62 fluidrachms) of pure hydrochloric acid mix enough urine to discolor this, then allow nitric acid to trickle along the sides and form a layer underneath. A beautiful play of colors takes place at the point of contact, and, on stirring up the mixture with a glass rod, throughout it.

The following is also a delicate test for bile. Add to the urine some calcium chloride solution, and then solution of sodium carbonate. The precipitate will contain any bile-pigment, and may be collected by agitating the liquid with chloroform. The chloroform solution

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[1] Garrod, Med.-Chirur. Trans., 1899.

[2] Diseases of the Liver, Sydenham Soc. Transl., vol. i. p. 100.

[3] Clinical Lectures on Diseases of the Liver.

should be agitated with water and acidulated with acetic acid. Any bilirubin will color the chloroform yellow, which will become green on adding the acid.

If the urine contain only altered biliary coloring-matters (bilifuscin), they may, according to Hofmann and Ultzmann, be recognized as follows. A piece of clean white linen is dipped into the urine, and then allowed to dry; it is discolored brown. Further confirmation is found in a very dark reaction for urophæin (by adding about double the quantity of urine to strong sulphuric acid), the urine appearing not garnet-red, but black. A similar reaction is produced only by the presence of sugar and of blood-coloring matter, both of which can be excluded by the appropriate tests.

The *biliary acids* are sought for by *Pettenkofer's test.* It consists in adding a few drops of a solution of sugar to a small portion of urine contained in a test-tube or in a china dish, placed in cold water. To this mixture an excess of concentrated sulphuric acid is added, drop by drop. The fluid assumes a yellowish-red color, which, if bile be present, passes into a crimson or violet. But it is inconclusive; for urine containing an excess of indican or oleic acid or albumin may display, when thus treated, a reaction similar to that caused by the bile acids. The spectrum, which shows lines by F and near to E, affords, according to Schunck, the most certain test of bile acid; indeed, minute distinctions between the different coloring-matters cannot be attained except through spectroscopy.

A delicate test very generally used for biliary acids is *Oliver's test.* The test solution consists of half a drachm of pulverized peptone, four grains of salicylic acid, half a drachm of acetic acid, and distilled water to make eight ounces. The fluid is made transparent by repeated filtering. Twenty minims of urine are added to sixty minims of the test solution; if bile acids are in excess, a distinct milkiness quickly appears.

Indican.—Among the so-called ethereal sulphates occurring in urine, of special significance is potassium indoxyl sulphate, indican. It exists in mere traces in normal urine. A notable increase in amount is regarded as evidence of increase in intestinal putrefaction. It is also found in all wasting diseases, and in morbid states attended with rapid decomposition of albuminous substances, as in empyema. It has been particularly noticed in obstinate constipation and obstruction of the small intestine. Occasionally the blue color of indican may be observed in urine soon after it is passed. Indican may be detected by the following test:

Add to a sample of the urine an equal volume of strong hydro-

chloric acid, and then a few drops of a solution of chlorinated soda. A bluish-black cloud is formed just beneath the surface of the liquid, and on stirring the reaction takes place throughout the mass. If the liquid be shaken with chloroform, the color will pass into the chloroform and collect at the bottom of the tube. Care must be taken not to use much chlorinated soda. The depth of color gives an approximate idea of the amount of indican present.

Sugar.—This substance is not a normal ingredient of urine, or exists only in traces too minute to be detected by the ordinary tests. When met with in normal urine it is probably due to the decomposition of the indican. Sugar may be found occasionally in the urine of those who live exclusively on a starchy diet, or who take large quantities of sugar; but the proportion even then is very small. It may also form from the breaking up of albuminous substances. Sugar appears in the urine after inhalation of carbon monoxide, and, as this is a common ingredient in illuminating gas, cases of light chronic poisoning giving rise to apparent slight diabetes are probably not uncommon. The urine secreted while under the influence of turpentine, ether, chloroform, chloral, or amyl nitrite is found to respond to the copper tests for sugar. Bordier[1] has grouped together many observations which led him to conclude that saccharine urine may be considered as an almost normal occurrence in the stage of recovery from acute diseases. Measles, pneumonia, erysipelas, all inflammatory fevers, are likely to exhibit it during convalescence. It may be detected in certain lesions of the brain and spinal cord and in phthisis. But a large and persistent amount occurs only in diabetes.

Urine holding sugar in solution is light-colored, of high specific gravity, and of peculiar smell. It rarely deposits sediments, and the excess of water in it may be large.

To detect the presence of sugar, several tests have been proposed, nearly all of which are easy of application. When albumin is present, this should be first separated by boiling and filtering.

Trommer's Test.—A few drops of a solution of copper sulphate are dropped into the test-tube holding the urine. Solution of caustic soda is now added in excess. If the fluid be saccharine, the faint greenish tint is changed to a deep blue, the precipitate which is formed when the alkali is first added being soon redissolved. On heating the blue mixture it becomes brownish, then yellow, and finally a reddish-brown mass of copper suboxide is thrown down, very different from the flocculent or greenish sediment noticed when no sugar exists. A

[1] Archives Générales de Médecine, 1868.

very small quantity of sugar can be detected by this process; but, good as the test is, it has its drawbacks; for sugar is not the only substance which possesses the power of reducing the salts of copper. Chloral, cellulose, kreatinin, and to some extent uric acid and the urates, share with it this property. Furthermore, Beale has shown that the presence of ammonium salts will prevent the precipitation of the suboxide in urine containing but little sugar.

For the quantitative determination of sugar, Fehling's solution is generally employed. This may be made by the following formula, in which, in accordance with the recommendation of Allen, the quantity of Rochelle salt is rather greater than ordinarily given. 34.64 grammes of pure crystallized copper sulphate are dissolved in pure water, and the solution is made up to 500 cc. 70 grammes of caustic soda in sticks and 180 grammes of pure Rochelle salt are dissolved in 400 cc. of water, and this solution also is made up to 500 cc. The two solutions should be kept in separate well-stoppered bottles. For use equal quantities are mixed as required. To determine the proportion of sugar in a sample, five cc. of each solution are mixed, diluted with about an equal volume of water, and brought to the boiling-point, in a porcelain basin. The porcelain dish with handle, called a casserole, is very convenient for this purpose. No precipitate nor loss of color should result from the boiling of the solution. The sample of urine is then added by small portions at a time, boiling between each addition, and watching the liquid so as to note the point at which all the blue color is removed. The condition is best determined by withdrawing the basin from the flame from time to time, inclining slightly, and allowing the red precipitate to settle. Any trace of blue color is easily seen. Ten cc. of the solution require .05 gramme of glucose to reduce them completely; the amount of urine used, therefore, contains this amount of glucose, and a calculation of percentage can easily be made. To get accurate results, the urine should be quite dilute, and if the qualitative tests indicate considerable sugar it will be necessary to dilute the liquid to five or even ten times its bulk. This dilution must, of course, be allowed for when making the final calculation.

Allen recommends the following test for cases in which there may be doubt as to the presence of sugar. Heat, to boiling, about ten cc. of Fehling's solution, and add a nearly equal quantity of the urine; heat for a few minutes, and then set aside to cool. If no turbidity is produced as the liquid cools, the urine is free from sugar, or, at most, contains less than $\frac{1}{40}$ per cent. Fehling's test can also be used for peptone and propeptone. It gives at the point of contact in the test-tube a rose-pink or purple color.

Boettger's Test.—Add to the filtered urine about half its volume of sodium hydroxide solution and a pinch of pure bismuth subnitrate, and boil the mixture. Sugar will be indicated by a *black* precipitate. If sugar is not present, the precipitate will be white, or, at most, somewhat gray. This test is very delicate and tolerably free from fallacy. Dark-colored urines of high gravity may produce a gray precipitate, but it does not settle so rapidly nor so completely to the bottom of the tube. Only a pure, finely powdered preparation of the bismuth compound should be used for the test. The bismuth test has an additional value, because alcaptone in the urine, which reduces the Fehling test and thus leads to the mistaken idea of the presence of sugar, does not influence it.[1]

Phenylhydrazine Test.—Phenylhydrazine is a coal-tar derivative which possesses the property of forming crystalline compounds not very soluble in water with bodies of aldehydic or ketonic type, to one or the other of which classes the sugars belong. It is generally used in the form of phenylhydrazine hydrochloride. It is said to cause a persistent eczema when much in contact with the skin. The test may easily be conducted without danger. The following method seems, according to some comparative experiments made by Leffmann on the different published processes, to be the best. Fifty cc. of the urine are mixed with 0.75 gramme of phenylhydrazine hydrochloride and 1.0 gramme of sodium acetate, and the mixture is heated for one hour at least in a test-tube placed in boiling water. Very small amounts of sugar will produce a marked yellow precipitate—a compound of sugar with the reagent—which under moderate magnifying power exhibits either brush-like branchings or more decidedly radiate crystals, somewhat like chestnut-burs. A flocculent brownish precipitate or small brown globules should be disregarded. The precipitate is almost characteristic of sugar, but cannot by the microscope be distinguished from a similar precipitate by glycuronic acid, a rare substance which is closely allied to dextrose in structure. The distinction can be made only by collecting the precipitate and determining its melting-point. The test, however, is principally of value in distinguishing those cases in which very limited reducing action is exhibited by a sample of urine when tried by the ordinary tests. As a delicate reaction for true sugar it does not seem to possess the great advantage over Boettger's test that has been claimed for it.

Further tests, though now not much employed, are *Moore's test*—boiling the urine with an equal part of potassium hydroxide—and the *fermentation test.*

[1] Futcher, Alcaptonuria, New York Medical Journal, 1897, ii.

Other forms of sugar, such as *sugar of milk*, may be found in the urine. Sugar of milk has hitherto been detected only in the urine of lying-in and of nursing women.

Acetone.—Ralfe gives the following test. About 4 cc. (one drachm) of sodium hydroxide solution containing a gramme (fifteen grains) of potassium iodide are placed in a test-tube and boiled. An equal volume of urine is then poured in cautiously, so as to float on the surface of the alkaline liquid. At the point of contact a ring of phosphates will be formed, and after a few minutes will be colored yellow and studded with crystals of iodoform. Alcohol and lactic acid also give this result.

On adding a very dilute alkaline solution of sodium nitroprusside to a fluid containing acetone, a ruby-red color is produced which in a few minutes changes to yellow.

Diacetic acid, a body somewhat similar to acetone, is occasionally present in urine. It is recognized by the red color produced by solution of ferric chloride in perfectly fresh, unboiled urine.

Both acetone and diacetic acid are derivatives of betaoxybutyric acid, and this itself results from the disintegration of the tissue albumins. Oxybutyric acid is now very generally regarded as giving rise to the acid intoxication that produces diabetic coma. In this it may be found in the urine in enormous amounts, 100 to 300 grammes in twenty-four hours.

The test for oxybutyric acid is with the polariscope. In thoroughly fermented urine, well filtered, the rays of polarized light are deflected to the left. For the quantitative examination duration is also necessary.[1]

[illegible] acid is formed by the direct oxidation of grape-sugar. The test for it is to boil urine with dilute sulphuric acid: the liquid polarizes to the right.[2]

Inosite.—This is a substance not belonging to the sugars, but having some of their properties, and at times found in the urine. Inosuria is a symptom rather than a disease.[3] The characteristic reaction of inosite is exhibited when a solution of the substance is evaporated with nitric acid nearly to dryness in a platinum, and the residue moistened with a little ammonium hydroxide and a solution of calcium chloride again evaporated to dryness: a marked rose-

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[illegible] and their [illegible] see Maly in Jahres Berichte, 1888.

[illegible] Med. Wochenschr., 1892, No. 27.

[illegible], Jahresb., 1864.

color appears,—which is not the case when true sugars are treated in the manner described.

The presence in the urine of the *blood-extractives* indicates merely the escape of blood-material, and proves the existence of congestion or inflammation of some part of the urinary surfaces. Rees has pointed out[1] that in Bright's disease the extractives can be found in the urine before albumin is met with, and also that they exist after the albumin has disappeared,—thus warning us, on the one hand, of the approach of albuminuria, and, on the other, against too early a belief in convalescence; for, as he justly observes, so long as the blood is losing its extractives so long is the patient in peril. The presence of the extractives also enables us to diagnosticate nephritic irritation from renal calculus before albumin, blood, or pus has appeared. To the delicate test by guaiacum for the crystalloids of the blood, which has been used to detect the prealbuminuric stage of Bright's disease, we shall presently more particularly refer.

Albumin and other Proteids.—The study of the various proteids occurring in urine is a matter of difficulty; and much uncertainty and confusion still exist with reference to them. The genito-urinary tract being a mucous area of great extent, abnormal secretions are frequent, and it is in many cases impossible to determine the boundary between health and disease. Thus, much discussion had been held as to the occurrence of albumin in *normal* urine, without any clear definition as to what is meant by the term normal. Efforts have been made to secure tests of extreme delicacy, but, while some of these have value in physiological investigation, they are often too delicate for practical work.

By the term albumin, unqualified, clinicians generally mean serum-albumin, and that meaning will be understood in this work. A proteid, derived from the mucous tissue, is generally present in urine. This has been designated *mucin*, but seems to be identical with a body called nucleo-albumin and also obtained from bile. Fibrin and hæmoglobin may appear, and also all the products of the transformation of proteids under the influence of digestive ferments, that is, the various proteoses and peptones; what is often designated peptone is an intermediate product,—an albumose.

Albumin appears sometimes for a short period and then for a time is not found. Egg-albumin, it is stated, may show itself in the urine after the free use of eggs as food.

The tests for albumin depend on coagulation. The most important are:

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[1] Guy's Hospital Reports, 3d Series, vol. xiv. p. 431.

Heat;
Nitric acid;
Picric acid;
Potassium ferrocyanide;
Trichloracetic acid.

Heat Test.—Albumin is coagulated by heat of about 150° F. (65° C.). The application of heat to normal urine often causes a precipitate of phosphate. To avoid this fallacy a small amount of acid, nitric or acetic, is added. The test is best performed as described by Purdy: Mix a portion of the sample with about one-eighth its volume of a saturated solution of common salt, filter, and fill a test-tube nearly full with the mixture. Add two or three drops of acetic acid, and boil the upper stratum of liquid. The contrast between the two layers of liquid will be sufficient to indicate very small amounts of albumin. The salt solution prevents the interference of mucin, which is not precipitated under these conditions. This is a satisfactory method for the detection of minute amounts of albumin. Small quantities may be also found by thorough boiling of urine to which a few drops of acetic acid have been added, without admixture with the salt solution.

Nitric Acid, Heller's Test.—Fifteen drops of commercial nitric acid are placed in a somewhat narrow test-tube, and some urine poured slowly down upon it, the tube being considerably inclined. Another method is to put the urine in first and introduce the acid by means of a pipette, so as to form a clear layer at the bottom of the tube. A white ring forms at the point of contact. Urine in which this test does not show albumin may be regarded, for practical purposes, as not containing it.

FIG. 62.

Albumin test-glass.

Tests by the so-called underlaying method are conveniently made by the use of the albumin-test glass designed by Kyner. The precipitating substance, *e.g.*, nitric acid, is put in proper quantity in the tube, and the liquid to be tested is allowed to flow through a filter folded in the usual way and placed in the funnel-shaped top of the glass.

In testing filtered urine it must be borne in mind that many forms of filter-paper will furnish enough soluble vegetable albumin to give distinct reactions with the more delicate tests for proteids. To avoid this error it will be best to use the centrifugal machine to secure a clear liquid.

Urine rich in urea sometimes forms a precipitate of urea nitrate. It may be distinguished from albumin by its crystalline character.

especially after standing a few hours, and by its solubility when the liquid is warmed. Excess of urates may also produce a precipitate that might be mistaken for albumin, but the ring is irregular and will in a few hours become distinctly crystalline and can be easily determined under the microscope.

Resinous bodies administered as medicines are precipitated by the addition of nitric acid. They may generally be recognized and distinguished from albumin by their strong odor and by their solubility in alcohol.

In urine containing alkaline carbonates an effervescence will occur when any acid is added, but this will soon cease and the coagulum will be formed. Convenient ways of determining the quantity of albumin in urine are by Esbach's albuminometer and Purdy's centrifugal method. The standard reagent for the former is composed of 10 grammes of picric acid, 20 grammes of citric acid, to 1000 cc. of distilled water. After admixture with the reagent, the urine must stand for twenty-four hours. With Purdy's electric centrifuge the test is more accurate, and can be completed in fifteen minutes. Graduated percentage tubes and acetic acid, and a solution of potassium ferrocyanide, are employed.[1]

Sometimes urine is encountered on which neither the heat nor the acid test yields the customary result. This is owing to its containing modified albumin, *albuminose*. Such a case was published by Bence Jones.[2] No coagulation was produced by heat, and none by nitric acid, unless the urine was subsequently heated and permitted to cool. The solid that formed on cooling disappeared on heating. The patient was suffering from mollities ossium. The test as now mostly practised consists in slowly heating slightly acidulated urine, which becomes cloudy, but clears on thorough boiling; on cooling, the cloudiness or the deposit reappears. Nitric acid produces in cold urine a deposit, which disappears on boiling, and reappears on cooling. A number of late observations, especially those of Kahler, Rosin, and Ellinger,[3] have associated this form of albuminose with multiple tumors of the marrow of the bones. In a case reported by Fitz[4] there was also myxœdema. Basham recommends the tincture of galls as a test for this modified form of albumin.

Picric Acid Test.—The saturated solution of this acid may be employed in the manner of the nitric acid contact test. The solution, being lighter than most urines, will form the upper layer. Picric

[1] Purdy, Journ. Amer. Med. Association, Sept. 23, 1899.

[2] Philosophical Transactions for 1848.

[3] Deutsches Arch. f. klin. Med., lxii., 3 and 4, 1899.

[4] Transactions of the Assoc. of Amer. Phys., 1898.

acid makes a very delicate test, but shows the same fallacies as the other acid tests. It also forms a slight precipitate with mucus, stains the skin yellow, and is somewhat explosive.

Potassium Ferrocyanide Test.—Twenty-five drops of strong acetic acid are thoroughly mixed with three times that amount of a solution of potassium ferrocyanide (1 in 20). A considerable volume of the urine is then added. Albumin if present will form a precipitate. The test thus applied is absolute evidence of albumin.

Trichloracetic Acid.—This is a solid, highly deliquescent body. It is corrosive, and should be handled with care. It is employed in the strongest possible solution, best obtained by allowing the solid to absorb water from the air until a solution is just formed. It is too delicate a test for general clinical work. It reacts with all proteids.

Halliburton sums up the reactions for the different *proteids* as follows:

If no precipitate forms on boiling after acidulating, albumin and globulin are absent. A precipitate may indicate both.

If no precipitate is produced after neutralizing the original liquid and saturating with magnesium sulphate, globulin and heteroproteose are absent. If the urine gives no precipitate by the boiling test for albumin, nor with nitric acid in the cold nor when saturated with ammonium sulphate, peptone is the only proteid that can be present. Peptone may be detected by the so-called biuret reaction, which depends on the red color produced by adding solution of sodium hydroxide to the liquid to be tested and then a small amount of a dilute solution of copper sulphate. Other proteids react with this test, but give a reddish-violet color.

The complete removal of all the other proteids from a mixture containing peptone by means of ammonium sulphate is difficult. Peptone is met with physiologically only during the puerperal state.[1] It occurs pathologically during many varying conditions, especially as the result of incomplete digestion and where there is tissue degeneration. It is frequent in general paralysis.[2]

Globulin very seldom occurs in the urine except in combination with serum albumin. But in advanced disease of the kidneys its relative proportion may be much increased. According to Senator it is most increased in waxy kidney. Estelle[3] met with a number of cases

[1] Robitschek, Zeitschr. f. klin. Med., xxiv.

[2] Arch. Gén. de Méd., March, 1894.

[3] Quoted by Hills, in a very instructive article on the Proteids of the Urine, Boston Med. and Surg. Journ., Aug. 1899, which may be also advantageously referred to for the relative study of the chemical tests.

in which globulin was the sole proteid. Globulin is insoluble in water, but soluble in dilute salt solutions.

Mucin, Nucleo-Albumin.—The reactions of this substance have been studied especially by D. D. Stewart.[1] He found that solutions of citric acid, both dilute and concentrated, used by the underlaying method, as in the cold nitric acid test for albumin, gave distinct contact rings. Picric acid associated with citric acid also gave such precipitates, but picric acid alone produced, with solutions containing not more than .02 per cent. of nucleo-albumin, only a tardily appearing haze. If urine be diluted with water and then strongly acidulated with acetic acid, mucin is precipitated, and may be collected, redissolved in water by the aid of alkali, and again precipitated by acetic acid.

Blood.—The passage of blood with the urine constitutes hæmaturia. The urine is of a red color, or of a smoky hue. If much blood be present, small, irregular masses are seen at the bottom of the vessel. But the only certain diagnosis is by the microscope; for urine may be red or black, from the admixture of various pigments derived from substances swallowed as food or medicine, or belonging to the economy. Thus, beet-root, some kinds of strawberries, logwood, and rhubarb impart a deep red color, which may be the cause of groundless alarm; or urine deeply tinged with bile, or discolored by fever, may be thought to signify the occurrence of hemorrhage.

The chemical tests for blood are much inferior to the microscopic examination. Yet we sometimes may have to resort to them. I have found a rough test in the addition of carbolic acid, which not only coagulates the albumin, but also changes the color of the fluid. It does not produce the same peculiar reddish tinge with bile, or, so far as I have tried, with any other substance. The guaiacum test is very accurate. It is especially valuable in the recognition of the pre-albuminuric stage of Bright's disease, in which hæmoglobin appears in the urine before albumin.[2] The test, as modified by Stevenson, consists in adding to a few drops of urine in a small test-tube a drop of tincture of guaiacum and then a few drops of ozonic ether. The mixture is agitated, and as the ether collects at the top it carries with it the blue color produced by the hæmoglobin, leaving the urine colorless below. If saliva or a salt of iodine be present, the test is fallacious. The spectroscope affords a very delicate test. The characteristic bands of hæmoglobin of yellow and green are seen between

[1] Medical News, July, 1894.

[2] Mahomed, Medico-Chirurgical Transactions, 1874.

D and E. If the hæmoglobin be in a state of destruction or reduction, only one broad band appears.

But the microscope, as already stated, is the means most employed and most valuable. The corpuscles we detect are often crenated, or very pale, and sometimes very small, but never collected in rouleaux; there is often considerable granular pigment. After having determined that hæmaturia exists, the questions remain to be solved, at what point has the blood been poured out? Is it really from the urinary organs? and if it be from them, whence?—from the kidneys, from the bladder, or from some other portion of the tract? Again, what morbid state lies at the root of the hemorrhage?

Now, the first of these questions must always be answered at the onset. Blood may flow from the vagina or uterus and become mixed with the urinary secretion, or it may have been added for purposes of deception. In the former case, a careful inquiry into the state of these organs, or, if necessary, a digital examination, will eliminate the source of error; in the latter, drawing off the urine by the catheter will detect the imposture. When we have fully satisfied ourselves that the blood is derived from the urinary organs, the next point to be ascertained is whether it proceeds from the kidney or from the bladder. To determine this, we have not only to study the character of the fluid excreted, but also to investigate all the conditions of the accident.

If the blood come *from the bladder*, it is not equally diffused through the urine; the fluid discharged is at first clear or nearly so, but at the end of the act of micturition is much more deeply colored; or pure blood, in a liquid form or in clots, is voided. Then, too, there is usually pain over the bladder, with a frequent desire to pass water, and a stoppage in doing so; the urine is generally alkaline.

When the blood is derived *from the kidney*, we mostly discover pain in the lumbar region, and other symptoms pointing to the affected organ, the existence of albumin in considerable quantities in the urine, or the passage of gravel. Clots are not encountered in renal hemorrhage, except when the blood coagulates in the infundibulum or the ureter and is gradually forced downward. Such clots are of a whitish color, and generally of cylindrical shape. In their passage towards the bladder and out of the urethra they become often the source of distressing pain. They are very significant, yet they are not absolutely pathognomonic of renal hemorrhage; for coagula formed in the bladder may be retained there for some time, and lose their color before they are expelled. Sometimes we meet with little solid or gelatinous fibrinous coagula which bespeak simply localized fibrinous exudation from some part of the urinary passages.

Aid in diagnosis may be derived from the study of the shape of the clots, which for this purpose should be floated out in water. According to Hilton,[1] they will oftentimes be exact moulds or casts of the cavity in which the blood was effused. Thus, coagula formed within the bladder have a somewhat irregular, circular outline, and are flattened in shape, with bevelled and serrated edges. The use of the microscope, furthermore, is very valuable in the differential diagnosis. The epithelium which is mixed with the blood from the kidney is not flat and in scales, like that from the bladder, but small and more or less round or columnar; nor are there fibrinous shreds. Sometimes the blood-corpuscles are observed to be collected on casts that have been moulded within the renal tubes. These blood-casts warrant an absolute conclusion as to the source of the hemorrhage.

Renal Hæmaturia.—When of renal origin, the hæmaturia is often due to congestion or an acute parenchymatous inflammation of the kidneys in infectious maladies, such as scarlatina, smallpox, malignant measles, and typhus. Here we have the history of the malady, and the presence of tube-casts, of blood-casts, and of a considerable amount of albumin, to explain the meaning of the hemorrhage. The blood is derived from the engorged and ruptured Malpighian corpuscles. It has been stated[2] as a diagnostic sign that in renal hæmaturia the blood-corpusles show fragmentation, similar to the irregularities of poikilocytosis, while this does not happen in vesical hemorrhage. But as regards the large amount of albumin present, we must not lay too much stress on this as indicating marked kidney implication. Irritant medicines, such as turpentine and cantharides, may cause congestion and bloody urine; and so do strains and blows on the back. In all these varied circumstances, a careful survey of the history and the symptoms will establish the diagnosis.

Renal hæmaturia of chronic character is generally due to cancer of the kidney; to cystic degeneration; to ulceration within the pelvis of the organ; or to irritation, with or without ulceration, set up by a calculus. In the first of these affections there is nothing in the urine to point out the source of the hæmaturia until the disease is far advanced, when pus, and sometimes disorganized cancerous tissue, may be discerned in the sediment. The manifestations of cystic degeneration are uncertain unless we can detect a large tumor; the signs of a non-calculous pyelitis are not definite, but hæmaturia is a rare symptom. The existence of a calculus—the most common of the causes

[1] Guy's Hospital Reports, 3d Series, vol. xiii. p. 19 *et seq.*

[2] Gumprecht, Deutsch. Archiv f. klin. Med., liii. 1894.

producing chronic hæmaturia—is indicated as the source of the hemorrhage by localized pain, leucocytes in the urine, and by the bleeding having followed active exertion, or a jar of the body from a fall, and by its recurring from time to time under circumstances like those just mentioned, favorable to the disturbance of a calculus lodged in the kidney. We find also hæmaturia in tubercular disease of the kidneys; as in cancer, it is apt to be intermittent. Hæmaturia is at times met with in interstitial nephritis. Then there is a form of hæmaturia unconnected with any obvious lesion, and apparently of neurotic origin, to which Klemperer and Harris have especially called attention.

Hœmoglobinuria, or *paroxysmal hœmoglobinuria*, as it is in its most marked form, differs from ordinary renal hemorrhage: the urine, although coagulable by heat and nitric acid, exhibits very few or no blood-corpuscles, but shows much granular pigment; there is blood dissolution, and only the blood coloring-matter is found in the urine: with the hæmoglobin is generally methæmoglobin. We may use the guaiacum test to develop the presence of the dissolved blood-cells; the hæmin crystals of Teichman can be produced, and with the spectroscope we find the oxyhæmoglobin bands between D and E, occasionally also the methæmoglobin bands in the red. The urine voided is generally of a deep blood-color, and within an hour or two, perhaps, changes suddenly to a pale straw-color. It shows an increased proportion of urea. According to Greenhow,[1] crystals of calcium oxalate are constantly passed during a paroxysm, and are absent at other times. The affection is unattended by any permanent lesion of the kidneys. It is paroxysmal in form, but not of malarious origin. It is ushered in by a chill; in some instances immoderate yawning and stretching of the limbs are the initiatory symptoms, and urticaria, great thirst, and local cyanotic appearances are observed. There is, indeed, a close association with Raynaud's disease. The temperature may be normal or elevated. Transitory albuminuria may precede the attacks; between them the urine is normal. Pain in the loins is not unusual. In the blood during the attack a marked diminution of red corpuscles is observed, as well as masses of granules and spindle-shaped bodies and other products of destructive change; and it is very likely, as Ponfick maintains, that the blood condition is primary and the hæmoglobinuria secondary: hæmoglobin in the blood-serum always precedes the hæmoglobin in the urine. The etiology of the disease is unknown. It often happens in syphilitic subjects. In those

[1] Transactions of the Clinical Society, 1868, vol. i.

predisposed, brain-worry brings on attacks; rest and food may prevent them. The influence of cold seems to be a very potent cause.[1]

Hæmoglobinuria also occurs in a non-paroxysmal form, as after extensive burns, or due to toxic causes, such as poisoning by chlorate of potassium, carbolic acid, naphthol, pyrogallic acid, salol, arseniuretted hydrogen. The poisons of the infective fevers, such as scarlet fever, typhoid fever, yellow fever, may also occasion it.

There is an intermittent hæmaturia which is malarial. This *malarial hæmaturia* may occur in daily paroxysms, or at longer but regular intervals. The bleeding sets in suddenly. The urine is albuminous, contains casts, hæmoglobin, and generally only few blood-disks; it shows a hæmoglobinuria rather than a hæmaturia. The attacks are mostly preceded by coldness of the extremities; elevation of temperature follows. When there are distinct fever and yellowness of skin, the hemorrhage from the kidney forms part of the disease known as hemorrhagic malarial fever, which will farther on receive more detailed consideration. Malarial hæmaturia is more common in men than in women.[2] It differs from ordinary paroxysmal hæmaturia above described in the greater regularity of the paroxysms, and in the influence quinine exerts on them, though by some quinine is regarded as the cause of the hæmaturia. Malarial organisms are especially found in the blood.

There is also a form of hæmaturia which is endemic and depends upon the presence of a *parasite*, Bilharzia hæmatobia. It prevails in the Mauritius, certain parts of Cape Colony, Natal, Egypt, and Brazil. The parasite inhabits mainly the small vessels of the mucous membrane of the urinary passages and the kidneys, and it gains access to these parts chiefly during the act of bathing in rivers. Persons affected with the Bilharzia hæmatobia are often observed to pass small renal calculi of calcium oxalate having for their nuclei the ova of this parasite;[3] they may also present chylous urine. A similar *parasitic hæmaturia*, due to the Filaria sanguinis hominis, is met with in India.

Further, there is a hæmaturia peculiar to infants. This has been described by Parrot,[4] under the name of *renal tubal hæmaturia*, and is

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[1] Rosenbach, Berlin. klin. Wochensch., 1880; Mackenzie, Lancet, Feb. 1884.

[2] Tyson, System of Pract. Med. by Amer. Authors, vol. iv.; see also Baker, Prize Essay, North Carolina Med. Journ., 1887; J. A. Stamps, Therap. Gaz., 1888, 3d Series, iv.

[3] Geo. Harley, Med.-Chir. Transact., vol. xlvii. p. 55, and vol. lii. p. 379; Handford, Brit. Med. Journ., 1887; Allen, London Practitioner, April, 1888, and Hill, London Lancet, May, 1888.

[4] Archives de Physiologie, Sept. 1873.

characterized by hæmaturia and the accumulation in the tubules of the kidney of the red globules of the blood, and by a bronze discoloration of the skin, and cephalic symptoms.

Besides these causes, renal hemorrhage may occur from rupture of the kidney, of which it is the most prominent sign. It may also result from an altered state of the blood, as in purpura and in scurvy or in leukæmia; or we may find hæmoglobinuria in these states.

Vesical Hæmaturia.—One source to which this may be owing is a congestion of the bladder, as witnessed in fevers of a low type; another is irritant diuretics; another is blood-effusion from purpura or the hemorrhagic diathesis. Yet another is inflammation, whether acute or chronic, and whether of traumatic origin or brought on by a stone. In most of these contingencies the history of the case and the local symptoms establish the diagnostic distinctions; in arriving at which we are often materially aided by the introduction of a sound into the bladder. In hemorrhage from the bladder, dependent upon tumor or malignant growths, there is generally also purulent urine; the appearance of blood in the urine may be the first sign of disease.[1]

Vesical hæmaturia, more frequently than renal, occurs as a vicarious discharge. Persons who are subject to bleeding piles lose blood occasionally from the bladder instead of from the rectum. But true vesical hemorrhoids are not uncommon.

Blood may be discharged from other parts of the urinary apparatus; it may come from the *prostate gland* or from the *urethra*. Now, in either case the bleeding is usually profuse, and large quantities of blood are passed pure, or unmixed with urine. Besides, the local signs furnish important points of discrimination.

Hæmaturia itself is very rarely fatal. One of the worst consequences it may entail is the retention of a clot which serves as a nucleus for the formation of a calculus.

Pus.—Urine containing pus deposits an opaque creamy sediment or a glairy mass, is generally alkaline, and always slightly albuminous. If the deposit be agitated with a strong solution of caustic soda it becomes gelatinous. This is the chemical test for pus. But it is a clumsy one, compared with the rapid and absolute diagnosis by means of the microscope. With the leucocytes we find considerable epithelium from the bladder or the pelvis of the kidney.

A deposit of phosphates may be mistaken for pus; a few drops of acetic acid clear it up, but do not influence pus. Sometimes a large amount of mucus is mixed with the purulent sediment, or a deposit

[1] See case by Todd, Case XI., Lectures on Urinary Diseases.

due wholly to the former ingredient is so considerable that it is mistaken for pus. Yet the mucous deposit shows distinct points of difference: it is less dense, and collects more in clouds at the bottom of the vessel; and it does not under any test show albumin. Again, the microscope is a valuable means of discrimination. In place of leucocytes, quantities of epithelium are always seen to be entangled in the transparent mucus, and the action of acetic acid develops the filaments of mucin. Sometimes, also, there are thin flakes of cylindrical bodies, unlike any appearance exhibited by pus. Yet, when the urine is strongly ammoniacal, even the microscope does not furnish a certain test; for the salts of ammonia obliterate the distinctive pus-globules

FIG. 63.

Leucocytes in the urine; those at the lower part of the field exhibit the action of acetic acid on the corpuscles.

and convert pus into a slimy mass, in which nothing but the nuclei may be distinguishable.

As to the exact seat of the formation of the pus, its existence in the urine affords no clue. When the leucocytes are round and well developed, with their characteristic nuclei readily brought out by acetic acid, they generally have their origin in a catarrhal inflammation of the mucous membrane of the bladder, and are apt to be associated with triple phosphates. On the other hand, pus-corpuscles of irregular contour, exhibiting irregular nuclei when treated with acetic acid, and very granular, partly destroyed cells, indicate the probable existence of deep-seated suppuration, ulceration, or tubercular disease; and in this we find also tubercle bacilli. The sudden appearance in the urine of large quantities of pus points to the bursting of an

abscess: an abundant deposit of pus in acid urine is chiefly noticed in pyelitis. In all instances we must be certain that the pus in the urine is not from a urethritis or a vaginal discharge. To be sure in the latter case the urine must be examined after catheterization.

Fat.—Fatty matter may occur in the urine in various forms and in different conditions. It may be found in the shape of globules, when oil or milk has been added to the urine for purposes of deception, or when the former article has been swallowed for some time in considerable quantities, as for instance during the administration of cod-liver oil. Fat is also encountered in globules of varying size, either free, in cells, or in tube-casts, as in fatty degeneration of the kidneys. Fat, too, may be found in the urine in cases of chronic suppuration, phosphorus poisoning, and in fat embolism after fractures.

The tests for fat are its solubility in ether, and its microscopical characters. Lee and Atlee have pointed out[1] an illusory detection of fat. They found, in testing a specimen of urine, that the ether rose to the top so charged with matter as to resemble a half-liquid pomade. Separated by a pipette and spontaneously evaporated, it left a dirty-white greasy mass. A careful examination of this residue showed that, instead of consisting of fatty acids, it contained nothing but the normal constituents of the urine, for it was soluble in water, reappearing as normal urine. It was then ascertained that almost any urine will form an emulsion when violently agitated with ether, especially if the ether contain a small amount of alcohol. When, therefore, ether appears to dissolve out fatty matter from urine, the ethereal solution should be separated, and allowed to evaporate spontaneously, and if the residue be soluble in water it cannot be held to contain fat.

There is no certainty of the presence of fat unless the sediment be examined chemically and microscopically. The opalescence of urine caused by a sediment of urates has been mistaken for that from oily matter, and so also has been the pellicle which often forms on urine, and which consists not of fat, but of vibriones, fungi, and crystals of the triple phosphates. The "kyestein" pellicle observed in the pregnant state is of similar kind, though some oily matter may enter into its composition.

In some cases fat is met with in a very finely divided state, imparting to the urine a milky look, which disappears on its admixture with ether. This condition, to which the name *chylous urine* has been given, does not depend upon any permanent morbid change in the

[1] Amer. Journ. Med. Sci., April, 1869, p. 357.

kidney; the chylous character of the urine is intimately connected with the absorption of chyle, but precisely how the urine acquires that character is uncertain. It may be absent in the day urine and very marked in the night urine; there are at times small quantities of albumin present. The affection may continue for years without impairment of the general health, being always perceptibly increased by exercise. In the tropics chylous urine is found often in connection with the Filaria sanguinis hominis.

A urine which spontaneously coagulates soon after being voided, owing to *fibrin*, a fibrinuria, is very uncommon except in the Isle of France and in Brazil. A thick urine may be due to pus dissolved in alkalies, as in certain bladder affections. But the thick matter is at once greatly thinned by water, and on the addition of acetic acid a white precipitate of alkaline albuminate falls.[1]

Sediments.—In connection with the ingredients of the urine, the nature of the urinary sediments has been discussed, and it has been insisted that they cannot be accurately determined save by a microscopical examination. I shall here group together only their general characteristics:

1. A light and flocculent cloudy deposit is commonly mucus, entangling epithelial cells, bacteria, or spermatozoa.

2. A dense, abundant, white deposit is generally composed of urates or phosphates; but it may be pus or extraneous matter.

3. A yellow or pink deposit is almost always due to urates.

4. A granular or crystalline deposit, of reddish or dark-brown color and small in quantity, is uric acid.

5. A dark, sooty or dingy-red deposit is blood.

6. A blue deposit is indican.

The following table may serve a useful purpose, in showing how both the sediments and the soluble urinary ingredients are affected by the reagents commonly employed:

TABLE EXHIBITING THE SIGNIFICANCE OF THE MAIN CONDITIONS AND THE ACTION OF THE MAIN REAGENTS EMPLOYED IN THE EXAMINATION OF THE URINE.

SPECIFIC GRAVITY.	High	Urine high-colored	Increase of urea, uric acid, etc.
		Urine pale	Diabetes.
	Low	Urine high-colored or normal	Certain forms of Bright's disease.
		Urine pale	Excess of water.

[1] Hofmann and Ultzmann, *op. cit.*

TABLE EXHIBITING THE SIGNIFICANCE OF THE MAIN CONDITIONS AND THE ACTION OF THE MAIN REAGENTS EMPLOYED IN THE EXAMINATION OF THE URINE.—*Continued.*

- **HEAT**
  - Throws down deposit
    - Soluble in nitric acid — Phosphates.
    - Insoluble in nitric acid — Serum-albumin. / Serum-globulin.
  - Dissolves deposit — Urates.
  - Does not dissolve deposit
    - Uric acid.
    - Phosphates.
- **NITRIC ACID**
  - Precipitates
    - Quickly — Albumin.
    - More gradually
      - Uric acid.
      - Urea nitrate (crystalline).
  - Dissolves
    - Earthy phosphates.
    - Alkaline phosphates.
    - Oxalates.
  - Produces play of color — Bile-pigment.
  - Turns black — Melanin.
- **HYDROCHLORIC ACID**
  - Precipitates — Uric acid.
  - Transforms
    - Urates into uric acid.
  - Change of color to violet — Uroxanthin.
  - To bluish / Admixed chloroform becomes bluish or violet — Indicans.
- **SULPHURIC ACID**
  - Changes color of urine
    - Brown — Urohæmatin.
    - Crimson or violet (if sugar have been added) — Biliary acids.
    - Violet — Indican.
- **ACETIC ACID**
  - Precipitates deposit (not soluble in excess of the acid) — Mucin (nucleo-albumin).
  - Precipitates with potassium ferrocyanide — Albumin and albumoses.

TABLE EXHIBITING THE SIGNIFICANCE OF THE MAIN CONDITIONS AND THE ACTION OF THE MAIN REAGENTS EMPLOYED IN THE EXAMINATION OF THE URINE.—*Continued.*

PICRIC ACID	Precipitates	Albumin, albumoses, peptones.		
		Red deposit,—blood.		
		Slowly-developed haze,—mucin.		
SODIUM HYDROXIDE	On boiling, turns urine brown	Sugar.		
	Dissolves	Uric acid.		
		Deposits of urates.		
	Forms gelatinous mass	Pus.		
AMMONIUM HYDROXIDE	Precipitates	Earthy phosphates.		
	Dissolves	Cystin.		
BARIUM CHLORIDE	Precipitates	Deposit, soluble in free acid	Phosphates.	
		Deposit, insoluble in acids	Sulphates.	
SILVER NITRATE	Precipitates	Yellow deposit, soluble in nitric acid and ammonia	Alkaline phosphates.	
		White deposit, insoluble in nitric acid, but soluble in ammonia	Sodium chloride.	
COPPER SULPHATE AND SODIUM HYDROXIDE	Precipitates with heat yellowish-red deposit	Sugar.		
	Turns violet	In cold	Peptone.	
		With heat	Serum-albumin.	
ETHER	Precipitates	Albumin.		
	Dissolves	Hippuric acid, soluble in alcohol.		
		Fat.		
	Does not dissolve.	Uric acid.		
BROMINE WATER	Turns urine yellow, then black.	Melanin.		

Toxicity of the Urine.—The human urine is toxic, and the toxicity varies under diet and in disease. The substances in the urine producing the poisonous effects are the potassium salts, phenol derivatives, unknown products of metabolism, coloring substances, as well as toxines obtained from various forms of bacterial infection. The toxicity is reduced by prolonged fasting and by a milk diet, although Lapicque and Marette [1] found that after the third day of exclusive milk diet it was again increased. The toxicity of human urine is reduced in anæmia;[2] the urine is also less toxic than normal in tuberculous lepers.[3] On the other hand, the toxicity of the urine is increased in cholera and in other infectious diseases, as well as in certain liver affections,[4] such as in atrophic alcoholic cirrhosis, in tuberculosis, carcinoma, some forms of chronic icterus, and in hypertrophic cirrhosis. It is normal or diminished in hypertrophic alcoholic cirrhosis, in conditions secondary to heart lesions, and in infectious icterus until the crisis, when it augments. Permanent increase is of grave prognosis, as it indicates destruction of liver-substance and function. The process of suppuration also increases the poisonous effect of the urine.[5] The toxicity of the urine is decreased in cases of puerperal eclampsia, whereas the toxicity of the serum of the blood is increased, as discovered by Bouchard, and confirmed by Ludwig and Savor.[6] The observation that the urine of epileptics is less toxic immediately preceding and during a fit, or series of fits, and hypertoxic after the attack, has been made by Voisin and Peron.[7] The urine of epileptics affected with mental disorder is also constantly less toxic. These observers therefore claim that by frequent estimation of the urinary toxicity it may be possible to predict the occurrence of a fit, to determine whether or not a series has terminated, or if mental disturbance is likely to follow.

The method pursued in order to determine the relative toxicity of the urine is to take a certain quantity of the mixed urine of the preceding twenty-four hours, filter it, and render it alkaline, precautions being taken to avoid bacterial contamination. The urine is then slowly injected into a vein in the ear or the leg of a rabbit or

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[1] Le Bulletin Médical, July 25, 1894.

[2] Piccini and Conti, Revue des Sciences Méd. en France et à l'Étranger, Paris, 1894.

[3] Chartinière, Annales de Dermatologie et de Syph., March, 1895.

[4] Surmont, La Semaine Méd., Paris, Jan. 20, 1892.

[5] Nannati and Baiocchi, Riforma Med., 1892.

[6] Monatshefte für Geburtsh. und Gynäk., 1895.

[7] Archives de Neurologie, Paris, 1892.

guinea-pig. Death of the animal follows after several ounces have been injected. By dividing the entire daily excretion of urine by the amount required to produce the lethal result (in cubic centimetres) and multiplying this by the fraction represented by the weight of the animal (in kilogrammes) as a numerator, and the weight of the patient as the denominator, a number is obtained which is called the toxic coefficient.

URINARY ORGANS.

Diseases of the Kidney of which Pain is a Prominent Symptom.

The group embraces acute inflammation of the kidney, and those painful affections classed under the term nephralgia.

Acute Painful Nephritis.—Acute inflammation of this kind is not a frequent disease, indeed, its very existence is not generally admitted; it is chiefly observed in old persons and in damp climates. It may be occasioned by exposure, by direct violence to the organ, or by the irritation of a calculus.

It begins with a chill, soon followed by fever of moderate degree; there are nausea and vomiting, and at times diarrhœa with tenesmus. The urine is voided drop by drop; it is red, and may contain blood. The patient complains of pain in the renal region, sometimes dull, at other times sharp and lancinating, and augmented by pressure and by moving. The pain is not limited to the kidney, but radiates to the diaphragm and to the bladder. With it are often associated numbness of the thigh of the affected side and retraction of the testicle. The disease rarely affects more than one kidney. It lasts from one to three weeks, and generally terminates in resolution. But it may lead to suppuration.

The disorder is recognized by the pain, the fever, the retraction of the testicle, and the appearance of the urine. It differs from an attack of colic by the signs of disturbance of the urinary organs, by the seat of the pain, and by the fever; from rheumatic pains in the back, by the former of these symptoms. Then, in lumbago, we rarely find much febrile excitement, nor are there nausea and vomiting, or numbness along the course of the anterior crural nerve; but, on the other hand, the pain is much more influenced by movements, especially by stooping, and such other motions as call the muscles of the back into play. Congestion of the kidneys is distinguished from inflammation by its affecting both sides, by the absence of protracted or severe pain, and by the comparatively slight derangement of the urinary functions. Further, the congestion is not idiopathic, and we

can generally trace it to the swallowing of some irritating substance, or to the poison of a febrile malady, such as smallpox or typhus. From the passage of a renal calculus acute painful nephritis differs by the steady, less paroxysmal and less violent pain, which does not, as in renal colic, begin suddenly and end suddenly; by the fever; and by the absence of a history of previous attacks.

Still, we must bear in mind that a calculus may be the cause of the painful nephritis. The distinction between this form of nephritis and that in acute Bright's disease will be presently considered.

Nephralgia.—Severe pain in the kidney, unconnected with inflammation of the organ, is ordinarily caused by the passage of a calculus. There is no fever, though passing elevations of temperature may occur. Nephralgia exhibits a great similarity to colic; but this has been already discussed; and in particular cases we are often much aided by the knowledge that in "renal colic" the patient has on a former occasion passed renal concretions.

The amount of pain varies according to the magnitude of the stone and its character. As a rule, calculi composed of *oxalate of lime* give rise to most pain. We may distinguish them by their roughness and irregularity and their brown or dark-gray color: those of *uric acid* and *urates* are reddish and much softer, and not jagged, and, unlike calculi consisting of the salts of lime, are combustible on platinum foil, leaving a mere trace of residue, while the oxalate of lime calculus leaves considerable residue, and is soluble in mineral acids without effervescence. Calculi of the *mixed phosphates* are white, very brittle, soluble in acids, insoluble in alkalies, and fuse in the blow-pipe flame. The mixed phosphates rarely form a stone entirely, being often only an incrustation around a blood-coagulum or a foreign body, or having a kernel of uric acid. Indeed, the majority of phosphatic stones have uric acid centres, while calculi of uric acid or its salts possess, as a rule, the same composition throughout; calculi of oxalates have often a nucleus of uric acid and a crust of phosphates. *Xanthine* and *cystine* are the rarer constituents of stones. The former, like uric acid and the ammonium and sodium urates, is consumed by heat, and burns without visible flame, but the murexide test exhibits an orange-yellow color; cystine burns with a bluish-white flame emitting an odor like that of burning fat, and the powder is soluble in dilute ammonia. The crystallization of the ingredients of the urine forming a calculus is very apt to take place around particles of mucus.

As already stated, we have in the severity of the pain a sign indicative of the nature of the case. Still, there are states in which *paroxysms of pain* referred to the neighborhood of the kidney are

attributable to other causes than the passage of a calculus. Leaving out of consideration that doubtful disease, pure *neuralgia of the kidney*, we find a few affections—very rare, it is true—which closely simulate the passage of a renal calculus.

The first of these is the pain occasioned by an *inflamed and ulcerated ureter*. Todd relates a case of the kind.[1] The patient had severe attacks of lancinating pain, referred to the right side, lasting for weeks, and accompanied by constant and intractable vomiting. The urine contained pus in varying quantity, but neither blood nor calculous matter could be detected. At one time he continued free from any paroxysm for four years. After death the most careful search was made for a calculus, but none could be discovered. The ureter of the right side was thickened throughout the greater part of its course, and deposits of lymph adhered to its mucous membrane. A somewhat similar train of phenomena may occur from irritation or inflammation of the ureter caused by the poison of rheumatism or gout, although the paroxysms of pain are apt to be neither so severe nor of so long duration.

Another morbid condition closely resembling the passage of a renal calculus may result from *malarial poison*. How close this resemblance may be, the following case will show:

A soldier, twenty-four years of age, of strong constitution, was seized suddenly with pain over the left kidney. The loin was sensitive to the touch, and appeared swollen. The skin was hot; the pulse 100. The urine was reddish, but was not found to be abnormal. The pain continued for several days, becoming more severe, notwithstanding that by direction of Dr. Hilborne West, with whom I saw the man, six ounces of blood were drawn from near the affected part. On the fourth day of the disorder he was assailed with excruciating pain along the course of the ureter, attended with the voiding, at short intervals, of a high-colored urine. The attack lasted from six o'clock in the evening until five o'clock the next morning, leaving him exhausted; the only relief throughout its duration being obtained from the inhalation of chloroform. At six o'clock that evening another seizure, of equal violence, set in; and, after the lapse of twenty-four hours, again another. Seeing the recurrence of the paroxysms at about the same time of each day, and learning from the patient that a few months before he had had a remittent fever, which had left behind an irregular intermittent, we resolved upon the administration of large doses of sulphate of quinine in the interval between

[1] Clinical Lectures, Lecture II., on Diseases of the Urinary Organs.

the paroxysms. The seizure did not take place that night; but, the remedy being a day or two afterwards suspended, the fourth night was again a night of anguish. The antiperiodic was resumed, and continued, in lessened doses, for three weeks. The patient remained under observation for about six weeks after the last attack, gradually recovering his health and spirits. When he was lost sight of, there was still a dull pain in the left lumbar region, with inability to stand erect; but no return of the excruciating intermittent pains.

In a case of this kind, which was observed before the days of Laveran's discovery, it is evident that nothing but a knowledge of the history of the patient, and the noting of the regularly recurring onsets of the pain, could have led to a correct appreciation of its cause. We sometimes meet with a so-called neuralgia of the bladder, of similar origin, and having much the same symptoms, except that the distressing pain is referred to the bladder. As in the case just detailed, the attacks occur at night.

These remarks are all based on the assumption that the renal pain is very severe and paroxysmal in its character. Let us now briefly inquire into the significance of a steady and less acute pain, premising that we have excluded from consideration abdominal aneurism, affections of the muscles of the back, of the spine, and of the tissues surrounding the kidney, in which diagnosis, of course, we are materially assisted by an examination of the urine.

We meet with *persistent pain* referable to the kidney itself, in inflammation of the organ, especially in that variety of inflammation affecting the infundibula and pelvis, termed pyelitis. We also encounter it in malignant disease of the kidney; sometimes, although it is not then of long duration, from the irritation of concentrated and highly acid urine; much more generally from the presence of a stone lodged in the kidney. The pain in the latter complaint often extends along the course of the ureter to the testicle, which is retracted and swollen. Not infrequently there is also tenderness on pressure over the affected kidney, and the pain is greatly increased by active exercise; and it is not uncommon to find, associated with these exacerbations of pain, nausea and vomiting, and the appearance of blood in the urine.

There is yet another point in the diagnosis of the *passage of calculi* which we must not overlook,—namely, that the pain may be referred to other parts than the region of the kidney and the course of the ureter. It may be felt near or at the sacrum, and not merely on one side; it may extend to the bladder and become associated with a painful spasm of this viscus and with the voiding of urine drop by

drop; or to the testicle, which becomes sensitive and swells; or to the thigh, which feels numb; or it may be referred to the region of the appendix, or to the right hypochondrium, and extend downward, but not be perceived in the loin. Under the latter circumstances there may be, with pain of great intensity, coexisting distention of the colon, vomiting, and constipated bowels, and the symptoms so closely resemble those of the passage of a biliary calculus that only the detection of blood in the urine prevents error.[1] Again, as happened in two cases which came under my notice, the pain may be referred to the left hypochondrium or along the course of the colon, may be associated with soreness to the touch and with digestive disorders, and may closely simulate an organic lesion of the stomach or intestine. Nothing but careful and repeated examinations of the urine, and observing the irregular and whimsical course the supposed intestinal malady pursues, will enable us to arrive at a knowledge of the truth.

Nor must we be unmindful that a calculus may be months in passing, and that as it changes its position the seat of the pain changes. I had a case of the kind under my charge in a lady about fifty years of age. She suffered for weeks at a time from excruciating pains, beginning in the left kidney, then felt somewhat below it, and finally localized in the neighborhood of the left ovary. She was occasionally free from pain for five or six days. But it was only after fully nine months of recurring suffering that the passage of a calculus the size of a plum-stone, followed by a discharge of large amounts of a gritty substance and a soapy-looking urine, removed her distress. The stone consisted of urates.

The symptoms of renal calculus may, after having existed for a longer or shorter time, entirely cease, owing to the calculus becoming encysted and thus remaining innocuous; or to its obstructing the ureter, causing retention of the urine, and, by pressure, producing gradual atrophy of the cortical and tubular structures, the kidney being finally converted into a mere bag.

In concluding the subject, it will be useful to group together the signs by which we may infer the existence of a *calculus in the kidney*. They are: frequent micturition, often attended with pain at the end of the penis; pain in the loin on one side, with or without accompanying soreness, occasionally passing suddenly into a violent paroxysm, with a tendency to shoot along the course of the ureter to the testicle and the hip of the aching side; and in some cases the dis-

[1] Case of Owen Rees, Guy's Hospital Reports, 3d Series, vol. x.

charge of pus due to coincident pyelitis. These symptoms become positive evidence if the blood-extractives be present in the patient's urine, or if this, when examined microscopically, be found to contain blood-corpuscles; or if we know that attacks of hæmaturia have previously happened, and that gravel or small urinary concretions have at any time been discharged. The presence, too, of microscopic calculi in the urine, points to the existence of larger concretions in the pelvis or in the structure of the kidney. But all these indications are far from being always present. The renal stones may be so large that they cannot leave the kidney; we may have nothing but the symptoms of a pyelitis, which we suspect to be calculous, and even these symptoms may be wanting. To determine whether both kidneys are implicated in the calculous disease, which occurs in about fifteen per cent. of the cases,[1] we must examine the urine during the passage of a renal calculus. If the urine become perfectly healthy, when previously it has been abnormal, we conclude that it comes from a healthy kidney, and that the secretion from the diseased one is temporarily blocked up. Another method of determining which kidney is diseased is by catheterization of the ureters, and the examination of the urine thus obtained from each. But this is a very difficult procedure, and is only possible in the hands of a surgical or gynæcological expert. Yet another method that has been suggested is by pressure on the pelvis, and the ingenious apparatus invented by Harris.[2] But the most certain of all our means is by the X-ray, and I add a reproduction of a skiagraph taken by Dr. Leonard from a patient in whom the calculus thus detected was removed by Dr. Keen. I have also seen a stone in the ureter brought to light by the same process. Irrespective of finding the stone, the Roentgen rays enable us to do what no other process can accomplish,—to detect the presence of several stones in the same kidney, and to determine their relative size and position.[3]

Diseases marked by an Albuminous Condition of the Urine, associated with more or less Dropsy.

The chief of these diseases is Bright's disease. At the present day we hold that the disease which bears Bright's name consists of a group of maladies having the common feature of a more or less albuminous state of the urine. But, though I believe this view to be the

[1] Henry Morris, Allbutt's System of Medicine, vol. iv.

[2] Journ. Amer. Med. Assoc., Jan. 29, 1898.

[3] Leonard, Phila. Med. Journ., Aug. 20, 1898.

PLATE IV.

correct one, I shall in this sketch prefer to consider the disorder in the main as it is seen separated by broadly drawn lines into an acute and a chronic form, and then examine the further differences these present. Anatomically speaking, we have a diffuse nephritis which is parenchymatous or interstitial; interstitial nephritis, generally seen in a chronic form, and often the result of gradual insidious tissue changes of a degenerative kind; the waxy or lardaceous kidney, and the fatty kidney, which is mostly an attendant upon other kidney alterations.

Acute Bright's Disease.—In this form, which is almost always an acute parenchymatous nephritis, the symptoms are of an acute character. Especially so is the dropsy, which is quickly developed

FIG. 64.

Epithelial casts and epithelial cells from the kidneys found in a case of acute Bright's disease (*acute parenchymatous nephritis*); magnified about 460 diameters.

and soon becomes the most marked token of the malady. The history of a large number of cases is as follows. After exposure to wet or cold and checked perspiration, a fever sets in, accompanied by nausea, and by a dull pain in the region of both kidneys, extending along the ureters. The eyelids and face become puffy and swollen, and soon a general œdematous condition of the skin is observable, showing itself very plainly in the extremities, scrotum, and abdominal parietes. Subsequently dropsical effusions often take place into the interior cavities.

The same symptoms are noticed in the acute parenchymatous nephritis, which so constantly attends scarlatina, except that, following as it does an exhaustive disease, there are from the onset much greater pallor and general debility. Acute parenchymatous nephritis is also met with, though less frequently, and generally in a less violent form,

in other infectious diseases, as in smallpox, measles, diphtheria, typhoid fever, typhus. It occurs also in malaria and yellow fever. It may follow hard drinking, a lightning-stroke,[1] or sewer-gas poisoning.[2]

The urine in the acute malady is of high specific gravity, and may be dingy from its admixture with blood. There is a frequent desire to void it, although the whole quantity passed is rather below the natural average. The urine contains a large amount of albumin; a microscopical examination brings to light red blood-cells and casts, lined here and there with blood-corpuscles. As the malady progresses, these "blood-casts" disappear, and we find casts coated with epithelium, which may be normal or slightly fatty, and with free nuclei; or we observe granular or hyaline casts; or we may discern leucocytes and long cylindrical ribbon-like mucous casts. Furthermore, crystals of uric acid, of urates, even of oxalates, and a considerable amount of renal epithelium, are often seen in the sediment. The chlorides and phosphates are diminished; the uric acid is less so, may, indeed, like the pigments, be increased. The amount of urea fluctuates much: it is generally lessened.

There is moderate fever, with a temperature of about 101°; the pulse, however, may be quick, tense, and full. The skin is generally harsh and dry; nausea and vomiting are of common occurrence.

The urgent symptoms last ordinarily for several weeks, and the albumin gradually disappears. But this is not the invariable issue; the disease may gradually lapse into a chronic form. Or a certain amount of albumin may remain in the urine; and after exposure this increases, and the dropsy and most of the acute symptoms return. In some instances of the malady, not in many, there are numerous tube-casts and free epithelium in the urine, but little albumin; and, on the other hand, in acute interstitial nephritis, with scanty, highly albuminous urine and marked general dropsy, tube-casts may be absent from first to last.[3]

There is a form of acute Bright's disease due to a bacillus. Letzerich[4] describes it as "nephritis bacillosa interstitialis primaria." It occurs in children, runs its course with a moderate fever in from two to six weeks, and generally ends in recovery. The urine contains red blood-corpuscles, a few leucocytes, only small amounts of albumin, but great numbers of bacilli, shorter and thicker than the tubercle

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[1] Medical and Surgical Reporter, July 23, 1887.

[2] Lancet, March, 1894.

[3] Dickinson, Allbutt's System of Medicine, vol. iv. p. 369.

[4] Neurol. Centralbl., 1887, quoted in Sajous's Annual, 1888, p. 483.

bacilli, and easily-stained with methyl-violet. An infectious nephritis also has been described due to the bacillus coli communis.[1]

Whatever the attending circumstances, the risk to life, when an attack of acute Bright's disease has been prolonged, is greatly increased by the supervention of local inflammations,—as of the pleura, lungs, peritoneum, or pericardium; or by the sudden effusion of fluid into the pulmonary structure; or by the retention of urea in the blood and consequent uræmic intoxication.

The recognition of the disease is readily effected. The puffy, pale face; the general dropsy; the albumin in the urine, associated with tube-casts,—form a combination of signs so remarkable that it is difficult to mistake their meaning. Many of the same phenomena are encountered in the chronic form of the malady; therefore, what is about to be said of the differential diagnosis of the acute complaint may be in the main applied with almost equal correctness to the chronic ailment.

The chief disorders with which acute Bright's disease is apt to be confounded are:

Acute Painful Nephritis;
Suppurative Nephritis; Purulent Urine;
Hæmaturia;
Simple Albuminuria;
Pulmonary Œdema;
Pleurisy and Pericarditis;
Dropsy;
Coma; Convulsions.

Acute Painful Nephritis.—This differs from acute Bright's disease by its affecting generally only one kidney, by the much greater pain and tenderness in the lumbar region, by the retraction of the testicle, and by the higher degree of febrile excitement. Then, too, the deeply colored urine which is voided contains little or no albumin.

Suppurative Nephritis; Purulent Urine.—In rare cases the suppurative process may coexist with Bright's disease. But, on the whole, the two disorders are distinct and may be readily discriminated. We find pus of renal origin in the urine, in consequence of pyelitis or of abscess of the kidney. The former is generally linked to the irritation of calculi, or is an infective process; the latter shows a fever of a remittent type, and often a well-defined swelling is felt in the lumbar region and extending far downward. All this is different from Bright's disease. Then, we detect pus as well as blood in the urine of cases

[1] Fernet et Papillon, Bull. et Mém. de la Soc. Méd. des Hôp., 1892.

of pyelitis or of renal abscess, and any casts that are found are apt to be covered with leucocytes, which is of very rare occurrence in acute Bright's disease.

Hæmaturia.—In hæmaturia, if we can speak of it as a separate disease, there is albumin in the urine; and, on the other hand, some blood as well as pus may be present in the urine of Bright's disease. But, as in purulent urine, the quantity of albumin met with in hæmaturia is small; in fact, it is in exact proportion to the amount of blood or pus the urine contains; whereas, on the contrary, if the secretion from a Bright's kidney be mixed with pus or blood, the amount of albumin is generally large. The microscopic examination, too, and the casts found, and their predominating character, are of great value.

Simple Albuminuria.—By this is meant an albuminous urine unconnected with any marked structural lesion, except congestion,—such an albuminuria as is observed as a transient phenomenon in the course of several diseases; as in the exanthemata, in typhoid, in typhus, in cholera, in hectic fever, in chronic congestion of the liver, in oxaluria, or as a consequence of surgical diseases and operations, and of ether narcosis. An albuminuria of similar kind is met with when the kidneys become congested from interference with the circulation, as in disease of the heart, or from the pressure of a gravid womb. Albumin in the urine may also be encountered in erysipelas, in diphtheria, in pneumonia, in acute rheumatism and in gout, consecutively to very high temperatures, to a burn, to a blister or a large mustard-plaster, or to the use of salicylic acid or of turpentine or of carbolic acid. But in all these conditions the quantity found is small and transitory, very unlike what it is in the persistent albuminuria of Bright's disease, and the urine is usually dense and high-colored. Then the constitutional symptoms and the general clinical features in the morbid states referred to tell us the meaning of the albuminuria. Moreover, there is really often more than mere congestion; there is present a parenchymatous inflammation to a limited degree, and of a transitory kind. In all these cases of albuminuria the amount of albumin is apt to be small, and there are few, if any, casts. When found, these are generally of the epithelial or hyaline variety, and are not highly granular or fatty.

In addition to these forms of simple albuminuria there is one of great importance to recognize, where the albumin happens in persons who in every respect seem healthy, and occurs shortly after partaking plentifully of food, especially of albuminous food, or after severe exercise, particularly in young persons at or near the age of puberty. Some of these cases are cyclic, occurring only at certain times of the day;

in much fewer, the albuminuria is persistent. In the great majority of cases there is a time in every day in which the urine is free from albumin. It is normal in quantity, normal or slightly increased in specific gravity, normal in the amount of urea it contains, and no tube-casts are found in its sediment. The amount of albumin in these functional albuminurias is small, and there are no cardio-vascular changes; indeed, there is no symptom except the albuminuria to suggest disease. This kind of albuminuria has a strong bearing on life assurance.

There is a form of albuminuria that stands in close connection with excessive uric acid formation and oxaluria, to which I have called attention.[1] The amount of albumin is generally small; hyaline and epithelial casts are found, though they are scanty. The specific gravity of the urine is high, and this, as well as urates or the oxalates in the urine, is of much significance. Violent exercise increases this albuminuria of uric acid and oxaluria. The cases may be of short or long duration; recovery is the rule.

In elderly people, we meet with a form of albuminuria in which there are traces of albumin in the urine, and hyaline and finely granular casts of small diameter. The specific gravity of the urine is normal, the general health is unimpaired. If this albuminuria be due to beginning senile changes, they are very slow in their development, and my experience leads me to the conclusion reached by the investigations of F. C. Shattuck, that it is of little practical importance.

Pulmonary Œdema.—Bright's disease is one of the most frequent causes of dropsical effusion into the air-cells; oppression in breathing, inability to lie in the recumbent position, cough, frothy expectoration, are the symptoms. And to distinguish this œdema from that produced by other morbid states we have only to examine the urine carefully. Yet we must not forget that small amounts of albumin may be found in the urine from any stress of breathing, and from diseases that, like those of the heart, congest the lungs and kidney and are themselves among the causes of pulmonary œdema.

Pleurisy and Pericarditis.—The tendency to inflammations of the serous membranes is a remarkable peculiarity of Bright's disease. We may discriminate pleurisy or pericarditis complicating the malady from either of these affections of other origin, by noting the far greater amount of dropsy that is found in these disorders, and by detecting persisting albumin and tube-casts in the urine.

Dropsy.—By an examination of the urine, too, may be distinguished the dropsy of the complaint under consideration from that

[1] Amer. Journ. Med. Sci., Jan. 1893.

produced by other causes. And we also see often the evidences of the true nature of the dropsy in its beginning with swelling of the face, and in the characteristic physiognomy which it has a share in developing.

Coma; Convulsions.—A dangerous complication of Bright's disease manifests itself by drowsiness and convulsions. Now, it is very important to distinguish the cases produced by uræmic poisoning from epileptiform convulsions and kindred states in which there is no appreciable change of structure in the kidneys. Let us see how they differ.

Uræmia, or uræmic intoxication, is commonly preceded by a diminution in the urinary secretion. In some cases the marked phenomena set in with a chill. There is headache, with indistinct vision, great drowsiness, and vertiginous sensations; the pupils are sluggish and usually dilated; the hearing is impaired; the countenance is dusky; the skin is cool, with short rises of febrile heat; and the patient suffers from constipation, nausea, and obstinate vomiting. Anæsthesia and various kinds of cutaneous eruptions may be observed. The dulness of mind is apt to deepen into stupor or coma, or convulsions set in as precursors of the coma, which terminates in death unless the urinary secretion be freely re-established. The coma may at one time be so profound that it is impossible to arouse the patient, whilst at another time he rouses himself and acts with intelligence. The convulsions generally succeed one another rapidly.

As regards the decided lessening, or suppression, of the urinary secretion, though this is the rule, it is not constant. I have known the symptoms of uræmia many a time to receive an erroneous interpretation, from supposing that uræmia could not exist, as the quantity of urine passed was about normal. We must test for urea and the other urinary ingredients, which may be profoundly changed in amount, notwithstanding the seemingly healthy aspect of the secretion, and notwithstanding, too, that it may be found free from albumin. In addition to the great decrease in the urea, the uric acid is reduced; the specific gravity is generally low; casts are mostly found in the urine.

Cases of uræmic coma differ from ordinary comatose conditions, as witnessed in apoplexy, in fevers of a low type, or following narcotic poisoning, by the dissimilar symptoms ushering them in. The coma is much more suddenly developed than that in fevers; far less suddenly than that of apoplexy or narcotic poisoning.[1] Then, the stertor-

[1] There may however, be exceptions to this rule, as in the case reported by Moore in the London Medical Gazette, 1845, in which a person became comatose after taking laudanum, yet his death was found to be caused by contracted kidneys.

ous respiration is peculiar:[1] the loud sounds of the expired air are of much higher key, not like the low, guttural tones of apoplexy. Furthermore, we may have in the general dropsy a clue to the nature of the case; but of course the most certain light is thrown on it by the analysis of the urine.

The same remarks apply to the delirium or to the epileptiform convulsions of uræmia. Here the difficulty in diagnosis is increased by the first seizure often happening unexpectedly,—so much, in truth, increased, that, unless we are aware of the history of our patient and have previously examined the urine, the true explanation of the symptoms is not to be reached. *Urœmic delirium* is rare, but I have met with it under circumstances in which nothing preceded it to indicate its nature.[2] Cases of *acute urœmic mania* may also originate thus suddenly. Cases of *urœmic convulsions* may occur in pregnant women; in them, however, the tendency to disorder of the kidney is so great that we are rarely in error in concluding convulsions to be of uræmic origin. We must, however, here, as in all convulsions, be certain that we do not mistake effect for cause. A slight amount of albumin may follow violent convulsions in epileptic seizures. The temperature in uræmic convulsions is variable. It is generally stated to be low; but this is denied by Bartels, who notes it as considerably elevated,[3] and by McBride,[4] and by Hughes.[5] Among the other marked nervous manifestations of uræmia may be persistent headache, anæsthesia, temporary blindness, and palsies of uræmic origin, local or hemiplegic, without gross lesion in the brain.

Uræmia is sometimes a chronic state, more particularly in chronic interstitial nephritis. Any of the symptoms already described may be met with; very common are nausea, vomiting, dyspnœa, headache, and eye disturbances. Convulsions, too, epileptic in character, and either general or of Jacksonian type, are found, and in some cases stomatitis, in others a long-continued, though moderate, fever, often with considerable mental torpor.

The cause of uræmia is still undetermined: a contamination of the blood by retained poisonous urinary ingredients or poisonous substances that have formed from them always happens, though these toxines may be of different kinds.

[1] Addison, Guy's Hospital Reports, 1859.

[2] Case at the Pennsylvania Hospital, April, 1865.

[3] Ziemssen's Cyclopædia.

[4] American Journal of Neurology, 1883.

[5] Philadelphia Hospital Reports, 1893.

Chronic Bright's Disease.—An acute attack of Bright's disease may gradually pass into a confirmed malady, or the complaint may come on insidiously and develop itself slowly.

The transition from the acute to the chronic disease is indicated by the disappearance of blood from the urine, by its lessened specific gravity and the smaller amount of albumin it contains, by the temperature becoming normal, and not uncommonly by a temporary diminution of the anasarca and an increase in the quantity of urine voided. When the disease runs a more or less chronic course from the beginning, its initiatory steps are obscure. We generally find such cases in persons who are poorly fed and half clad, who live in damp, ill-ventilated houses, who are intemperate, or who have been subject to great grief or worry, or are saturated with malaria, or whose constitutions are ruined by syphilis or by scrofula, or who show signs of arteriosclerosis. The first symptoms noticed may be frequent desire to urinate; swelling of the extremities or of the face; increasing pallor and general debility; and headache, especially occipital headache. An examination of the urine reveals at once the cause of the protracted indisposition. Yet the renal disease may lead suddenly to a fatal termination without the patient having experienced any ill health. And even after the malady has been recognized, it is difficult to predict its course. We meet in many cases with the same phenomena as those of the acute variety, except the fever. But in others the signs are dissimilar,—the dropsy, for instance, is slight or is wholly wanting. The only constant and characteristic manifestations are the increasing anæmia, and the presence of albumin and tube-casts in the urine.

Where chronic nephritis is suspected the urine passed at different times of the day, especially the morning and evening urine, should be separately examined. Generally, the urine is of unchanged specific gravity, though this is lowered as the urinary solids and the urea are lessened. The albumin is variable in amount; its quantity may, indeed, fluctuate much in the same patient, and even change from day to day. It is persistent; yet it may disappear for a short time.

The tube-casts, too, are not uniform,—not nearly so much so as in the acute variety of the affection. We meet with hyaline casts, small or large; with casts besprinkled with shrivelled degenerating epithelium; with casts covered with granules or with oil-drops. In the progress of a particular case, nearly all these forms may be encountered, although, as we shall hereafter see, the preponderance of any one of them is of significance. There is only one kind we do not find in the chronic disorder; the one covered with well-developed epithelial

cells or blood-corpuscles. The apparent absence of casts from albuminous urine is not absolute proof of the non-existence of renal degeneration. In some cases their absence is only temporary, while in others they are small and few in number and easily escape detection. This is especially the case in the contracted kidney. In this disease methylene-blue is much more slowly excreted by the kidneys than in other forms of nephritis, or with normal urine.[1]

A great diversity of phenomena is thus witnessed in chronic Bright's disease, and the different grouping of the symptoms tells us to a very great extent the form of the chronic malady we are dealing with. But before considering its varieties let us, leaving out of consideration those affections for which both the acute and the chronic disease may be mistaken, and which have been already discussed, consider the conditions with which chronic Bright's disease in general may be confounded. They are:

Anæmia;
Neuralgia;
Chronic Rheumatism;
Chronic Bronchitis;
Asthma;
Disease of the Heart; Cardiac Dropsy;
Gastro-Intestinal Disorders;
Cancer; Tuberculosis; Cysts of Kidney;
Chronic Consecutive Nephritis;
Renal Inadequacy.

Anæmia.—There are few diseases which alter the blood so completely as chronic Bright's disease, and the gradual impoverishment of the waste-laden blood makes itself manifest by the increasing debility, and by the pallor and waxy look of the countenance. We may discriminate this well-marked anæmic condition from that unconnected with renal disease by the existence of albumin and tube-casts in the urine, and often also by the prominence of the dropsical symptoms. But it is essential to know that some of the phenomena —certainly albuminous urine and dropsy—may attend the anæmia following profuse or frequently repeated hemorrhages, without the structure of the kidneys having been impaired. It is difficult to distinguish these cases from true Bright's disease, except by taking into account the diminution of the albumin as the hemorrhagic tendency is lost, and the absence of tube-casts. The dropsy, unless it be considerable, can hardly be looked upon as a valuable differential index,

[1] Bard and Bonnet, Arch. Gén. de Méd., Feb. and March, 1898.

for a slight or moderate amount of dropsy, or even none, may be encountered in either morbid state.[1]

The ophthalmoscopic appearances presented by the retina afford help in distinguishing between the anæmia of Bright's disease and that produced by any other cause. Albuminuric retinitis is not limited to any form of Bright's disease. It generally happens in both eyes, and, though in the chronic variety of the malady it may greatly improve, it does not disappear. The sight itself deteriorates; and we have attacks of blindness, uræmic amaurosis, which come on suddenly and pass off suddenly.

Neuralgia.—This is not infrequent in the chronic form of Bright's disease. Neuralgia of renal origin may affect the fifth nerve, or other nerves; sometimes it takes the form of hemicrania, and it is often associated with disordered vision, or with impairment of other special senses; or it may coexist with persistent headache or with strange and anomalous nervous symptoms. Headache from Bright's disease may also be present without neuralgia; it may be of the nature of megrim, and occur in paroxysms attended with nausea and vomiting.

Chronic Rheumatism.—Frequently patients affected with chronic Bright's disease complain of muscular pains. The pain is dull, not increased on pressure; sometimes shooting, more like that ordinarily termed neuralgic. The pain is oftenest met with in those instances in which the dropsy is slight or wholly wanting, and an examination of the urine is then the only means of determining its real significance.

Chronic Bronchitis.—This is one of the most common complications of Bright's disease,—so common, indeed, that Rayer observed it in seven-eighths of his patients, and Wilks[2] states it, from an extensive analysis of cases, to have been more universal than any other single symptom, albuminous urine alone excepted. It is hardly necessary to add that the last-mentioned sign is the one that distinguishes this secondary pulmonary affection from all other forms of bronchial disease.

Renal Asthma.—Whether or not there be coexisting bronchitis, attacks of shortness of breath, like paroxysms of asthma, occur as the result of Bright's disease. This renal asthma is most common in the chronic contracted kidney. It has no features by which it can be recognized from ordinary asthma, except that the wheezing and the

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[1] The occurrence of marked albuminuria after hemorrhage, to which attention was here called, has been since studied by Fischl, Arch. f. klin. Med., Bd. xxix., by Quincke, *ibid.*, Bd. xxx., No. 4, and by others.

[2] Guy's Hospital Reports, 2d Series, vol. viii.

râles are not so marked, and that it does not subside by copious expectoration. It more resembles, indeed, cardiac asthma, and is most frequent at night.

Disease of the Heart; Cardiac Dropsy.—In very many cases of chronic Bright's disease there is disease of the heart, particularly hypertrophy. This manifests itself by the ordinary physical signs of hypertrophy. The vessels become more tense and rigid, especially in contracted kidney, and there is marked accentuation of the second sound of the heart. With these physical signs, dropsy may show itself, and in chronic parenchymatous nephritis frequently does so. With kidney and heart both affected, it is often difficult to say which is the primary lesion; either will occasion albumin in the urine.

Let us suppose that in cases of so-called cardiac dropsy we find albumin: is this a proof of coexisting Bright's disease? Not unless the amount of the abnormal ingredient be considerable, and more than occasional tube-casts accompany the albuminuria. Mere congestion of the kidneys, resulting as it does from an obstruction to the flow of the venous blood along the vena cava, may occasion albuminuria; but the presence of albumin is temporary, and its quantity small, and the specific gravity of the urine is generally high. A large amount of albumin, persistent and conjoined with characteristic tube-casts, shows that changes are present in the renal textures. When disease of the heart and disease of the kidney are combined, it is the disease of the kidney which generally produces the disease of the heart. The cardiac affection does not give rise to the renal affection nearly as frequently as supposed;[1] yet often both are the result of a common cause, as a general cardiac- and arteriosclerosis.

Gastro-Intestinal Disorders.—These are among the most usual consequences of the renal malady. They manifest themselves in various ways: by flatulency and indigestion; by diarrhœa; by nausea and vomiting. The latter symptoms are apt to occur when uræmic intoxication is developed. They may be, however, also met with at any period of the disease, and become so prominent as to throw into the background most of the other signs of the renal affection. I have seen cases of Bright's disease which first manifested themselves by apparently causeless nausea and vomiting; the tongue was clean.

The intestinal disorders may be due to submucous hemorrhage in connection with the changed vessels in granular kidney, and lead,

[1] See proof in Middleton-Goldsmith Lecture, 1888, on Relation of the Diseases of the Kidney, especially the Bright's Diseases, to Diseases of the Heart, by J. M. Da Costa.

as Dickinson has pointed out, to intestinal ulceration and perforation.

Cancer ; Tubercle ; Cysts of Kidney.—These morbid products affect the kidneys but rarely,—at all events, rarely in a form so marked as to give rise to conspicuous clinical phenomena. In all of them there may be albumin present in the urine, but it is generally in small amounts, and mixed with some ingredient having a more specific meaning. Thus, in *cancer* of the kidney we may find blood with the albumin ; indeed, hæmaturia is a very important symptom, and in some instances we discern with the microscope cells like those observed in any cancerous growth ; often the hemorrhages are profuse and frequently recurring, are preceded by severe pain, and we detect a palpable tumor in the flank, passing upward into the hypochondriac region and downward to the iliac region, or even forward, not affected by the act of breathing, and sometimes causing bulging posteriorly. In cases of melanotic cancer, whether it have its seat in the urinary apparatus or elsewhere, the urine becomes dark on exposure to the air ; there is melanuria. In children, cancer of the kidney is not a rare disease, and when we can exclude as the cause of the renal tumor cystic degeneration and hydronephrosis—in them congenital affections—we can diagnosticate the case with some confidence. In adults the diagnosis is always doubtful, at least when the disease is primary. A rapid and irregular growth of the one-sided renal tumor, severe pain, bloody urine, emaciation, and cachexia are the most certain signs. The disease is twice as common in men as in women. Sudden and rapidly growing varicocele is stated to be a symptom of malignant tumor.[1] *Syphilomata* of the kidney may be suspected from the history, but cannot be recognized with certainty ; they rarely cause pain or produce a tumor large enough to be detected, but mainly give rise to the ordinary manifestations of chronic Bright's disease,[2] most often of the amyloid form. At times syphilis of the kidney shows itself as an acute syphilitic nephritis. In *sarcoma* of the kidney the swelling in the abdomen attains, in children especially, very great size ; hæmaturia is comparatively rare, and the peripheral lymphatic glands do not become implicated.[3]

In *tubercle*, little yellow cheesy masses of degenerated tubercular matter may collect as a sediment, as in the cases referred to by Fre-

[1] Guillet, Tumeurs malignes des Reins, Thèse de Paris, 1888.

[2] Wagner, Archiv f. klin. Med., Bd. xxviii., 1881 ; Mauriac, Arch. Gén. de Méd., Oct. 1886 ; Jaccoud, Gaz. des Hôp., 1888.

[3] Neumann, Archiv f. klin. Med., Bd. xxx., 1882.

richs in his work on Bright's disease. The tubercular matter is generally derived from the pelvis of the kidneys. With the albumin, pus and other signs of chronic pyelitis are present. The disease may be primary, or the infection take place from the bladder, the prostate, or the ureters. We may be assisted in the diagnosis by finding tubercles in other organs, as in the lungs; or there may be scrofulous disease of the vertebræ. In tubercle of the kidney, extreme pain, occurring in paroxysms like those of nephritic colic, is a very important sign. This pain, as I have had occasion to observe, is associated with frequent micturition, and is temporarily relieved by the flow of water. The urine is, however, scanty, and generally of low specific gravity. A moderate amount of hæmaturia may happen; tube-casts are rare; the patient passes at times little fibrinous shreds, has irregular fever, and emaciates steadily. The bacillus of tubercle in the urine serves as a means of diagnosis. In some cases the kidney most diseased enlarges sufficiently to form a tumor discernible through the abdominal walls.

In *cysts* of the kidney—those at least enclosing echinococci—vesicles containing the characteristic structures of the parasites may be perhaps detected. Ordinary cysts, when small, are not to be recognized with any certainty during life: nor can they be distinguished from Bright's disease; they are, indeed, frequent in the chronic varieties of this disorder. When the cysts attain decided dimensions, they give rise at times to the discharge of highly bloody urine, and to albuminuria, and to large tumors, which may be detected through the front walls of the abdomen. They may affect one or both kidneys, producing slow cachexia and enormous abdominal swelling. Cysts of the kidney and liver often coexist.[1]

Chronic Consecutive Nephritis.—In consequence of affections of the bladder, of stone in the bladder, of strictures of the urethra, of disease of the ureters and of the prostate, indeed of various surgical affections of the urinary organs, we may have a kidney disease established which is rather a form of slow inflammatory change than Bright's disease. It may affect only one or both kidneys, and the diseased organs are tough and hard, large or small, and show great increase of fibrous tissue. The source of irritation which has led to the secondary inflammation is at times in the kidney itself, in the shape of a large calculus in the pelvis.

In another form of this consecutive nephritis suppuration takes place, affecting first especially the pelvis of the kidney, a suppurative

[1] Sabourin, Arch. de Phys., ix., 1882.

pyelonephritis,—the condition often called *surgical kidney*. It is difficult to distinguish these consecutive forms of nephritis, especially where pus is found in the urine, either from the condition last mentioned or from coexisting bladder disease, except by the history. Very often there is pain along the course of the ureter; and the urine, when passed free from pus, contains neither albumin nor casts, or only a small amount of albumin and a few hyaline casts. The urine is apt to be copious and of low specific gravity. When it contains pus from the kidneys,

FIG. 65.

Fatty casts and epithelial cells filled with fat, as seen in the discharge coming from a highly fatty kidney.

and the bladder is comparatively unaffected, the purulent urine is generally acid. The heart rarely becomes disturbed, though hypertrophy has been occasionally noticed in the non-suppurative form.[1]

Renal Inadequacy.—There are patients who pass the ordinary amount, or less than the ordinary amount, of urine daily, of low specific gravity, from 1002 to 1008, not containing more than two per cent. of urea, though the uric acid may be normal, and who in consequence of this insufficient action of the kidneys are always ailing and weak, take cold easily, and suffer from headache and nervousness. Even if they drink water freely, they do not pass more urine; this does not contain albumin or casts, differing in this respect from Bright's disease. But dropsy, as Sir Andrew Clark, who first described the complaint,[2] states, with puffy face and dry, glossy skin, may happen, and a state similar to myxœdema be gradually developed.

Having now treated of chronic Bright's disease as *one* affection, I

[1] Fagge's cases, in Practice of Medicine, 1886, vol. ii. p. 483.

[2] British Medical Journal, vol. i., 1883.

shall briefly refer to the distinctions between its forms. In so doing, I shall follow the classification based on the diversified anatomical aspect of the kidneys.

First there is the chronic *enlargement* of the organ, of which several kinds exist:

1. *The enlarged chronically inflamed kidney*, known also as the large white kidney, or as chronic parenchymatous nephritis. This variety of the malady may or may not be preceded by acute nephritis. It may last for a few years, but generally terminates fatally before

FIG. 66.

Hyaline or waxy casts, magnified about 460 diameters. On some of them are scattered a few shrivelled epithelial cells and oil-drops; the large cells to the left are epithelial cells from the bladder.

The kind of casts here depicted may be found in any form of Bright's disease, acute as well as chronic. In the waxy kidney, however, they vastly preponderate, and are of large size,—many much larger than those in this figure.

that time. The urine is diminished in urea and pigment and in chlorides; it contains large amounts of albumin and granular and epithelial casts, with some hyaline casts and a few slightly oily casts. The dropsy occasioned is extensive and persistent, and there is usually little difficulty in tracing it to an acute attack. Sometimes the dropsy lessens materially, then actively recurs. The large kidney rarely contracts; but it may do so. The large white kidney may also pass into the fatty kidney. Dilatation of the heart is common in chronic parenchymatous nephritis, more common even than pure hypertrophy, which is more usual in contracted kidney.

2. *The fatty kidney.* The kidney is very large and fatty. The convoluted tubes are filled with oil, accumulated in their epithelial cells. The fatty disease is recognized by the numerous oily casts, fatty cells, and free oil-cells which appear in the highly albuminous urine.

It is a fatal complaint, generally very chronic in its course, and attended with persistent dropsy. This morbid condition must not be confounded with a simply fatty kidney, such as is sometimes found in phthisis or oftener in drunkards, and which is not associated with albuminous urine. A certain amount of fatty casts and fatty cells may appear in the urine and not be persistent or indicate the real, dangerous fatty kidney. Acute nephritis from cold and exposure is much more apt to be followed by fatty kidney than the acute nephritis attending scarlet fever, which is more likely to pass into the large white kidney. A fatty kidney is sometimes combined both with the granular and with the lardaceous kidney.

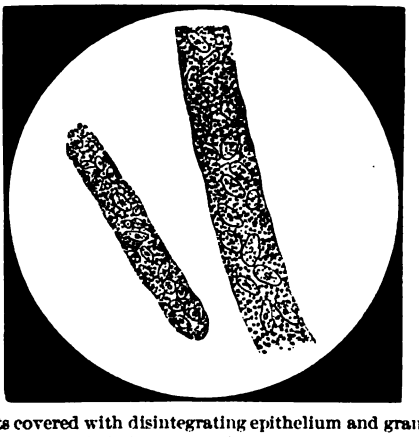
3. The *waxy* or *amyloid kidney* is the result of a general lardaceous or waxy disease involving the kidneys in common with other organs, and generally following upon protracted suppuration from any cause, either wound or disease. The urine is increased in quantity in the earlier stages, and of low specific gravity; it contains much albumin, but not many casts. Those which are seen are pale, and for the most part hyaline, or highly refracting, structureless moulds of the tubules of large diameter; they may or may not give the characteristic amyloid reaction, the red color when treated with a watery solution of iodine and of potassium iodide. Methyl-green colors amyloid substances an intense green. It is used for staining in the form of a one per cent. aqueous solution. Methyl-green colors hyaline casts *in situ* ultramarine blue, so that these also can be readily distinguished in sections of the kidney from the green-colored tissues around, in which they may lie. Blood is rarely present in the urine of the amyloid kidney, and the urea is but slightly diminished in quantity. Diarrhœa frequently coexists, and the liver and spleen are apt to be enlarged: but the heart is not affected. The dropsy is absent or trifling in amount, yet its persistence while the urine is increased in quantity is peculiar to this form of renal disease, and it may exist markedly as a late symptom; the patient is sallow-looking and emaciated; his disease may last for years.

In laying stress on the hyaline and waxy casts we must be careful not to confound them with those still larger mucous moulds of the uriniferous tubules, or *mucous casts*. They are also smooth, but of enormous length, subdividing into smaller ones, and of cylindrical shape. They are met with in acute parenchymatous nephritis, but occur particularly in consequence of transmitted irritation from the bladder, and are then associated with small amounts of albumin and of pus. Yet unless the latter be present there is no albumin, or the merest trace. Further, flask-shaped hyaline bodies and cylinders

may be moulds of the vesicles and smaller ducts of diseased prostates.[1]

4. Then we have the small *contracted kidney*, the granular kidney, or interstitial nephritis or renal cirrhosis. This form of disease is frequently found in gouty persons, in alcoholics, or after prolonged mental anxiety and distress, or in connection with general arteriosclerosis, or as the result of lead poisoning. The urine shows but an inconsiderable amount of albumin; the tube-casts are granular, or simple fibrinous moulds, generally small, sometimes large; here and there

FIG. 67.



Granular casts, or casts covered with disintegrating epithelium and granules. Casts of this character are chiefly found in the chronic inflammatory forms of Bright's disease, both parenchymatous and interstitial. The granular matter may be coarse and dark.

a litfle oil is observed. But though the urine may contain only small amounts of serum-albumin and of globulin, there may be a considerable quantity of other proteid matter in the shape of albuminose.[2] Dropsy is absent in a certain proportion of cases, and when present is generally slight. It often disappears for a while and returns. The urine is increased in quantity, although towards the termination it may become scanty or even suppressed. Dyspepsia, puffy eyelids, chronic bronchitis, increased arterial tension, hypertrophied ventricles, albuminuric retinitis, headache, and disorder of the nervous system are common symptoms. The malady runs a very chronic course. It is chiefly characterized anatomically by an affection of the fibrous tissues surrounding the Malpighian corpuscles and lying between the tubes, a slow increase, followed by a slow contraction, of the inter-

[1] Sir Andrew Clark, Transactions of the Clinical Society of London, vol. xix., 1886.

[2] Rose Bradford in Allbutt's System of Medicine, vol. iv. p. 304.

tubular fibrous tissue and atrophy of the tubules, connective-tissue changes in the renal plexus,[1] and fibroid changes in the small vessels of the body. The sphygmograph shows marked pulse-tension, and this, with altered specific gravity, has been noticed before albumin is present in the urine. In the uric acid or gouty nephritis, uric acid deposits may be found in the straight tubes of the medullary substances. A chronic interstitial nephritis may be also associated with deposits of lime, which take place very generally in the uriniferous tubules in the cortex. These lime deposits may be, as Virchow points out, calcareous matter washed into the kidney from diseased bone.

In contracted kidney, especially in the earlier stages, albumin, even casts, may be absent from the urine, and we may have to recognize the malady rather by the hypertrophied heart and thickening of the vessels, the high arterial pressure, the accentuation of the second sound of the heart, the headache, vertigo, nausea, breathlessness, retinal changes, and the anæmia. The urine may be of low specific gravity and copious, but there are many exceptions to this; it is generally deficient in urea. A few hyaline or granular casts are at times found: and the albumin may not be entirely absent, but appears every now and then in traces. There may be even chronic general œdema present without albuminous urine,[2] and various nervous and mental symptoms.

Stewart[3] has called attention to cases of chronic granular kidney without albumin, though generally with hyaline or finely granular casts, and with cylindroids, but with habitual diminution in the amount of urine and of the urinary solids, especially the urea, and with symptoms of retention of nitrogenous waste. Among these, debility, headaches, and vertigo are very prominent; there are no cardio-vascular changes.

Cases of fibroid kidney following generalized arteriosclerosis cannot be distinguished from primary granular kidney, except by the history of previous organic change in the heart and blood-vessels. Nor is the distinction of any importance. Chronic interstitial nephritis may be wholly latent, and nothing but an attack of endocarditis or pericarditis, or apoplexy, or convulsions call attention to its existence.

The different kinds of albumin have been above mentioned. Of these serum-albumin and serum-globulin are by far the most important, and have much the same clinical significance. With reference to the tube-casts, no special kind is of diagnostic value; it is the preponderance of the type alone that is. Hyaline casts have the least significance.

[1] Da Costa and Longstreth, Amer. Journ. Med. Sci., July, 1880.

[2] As in Case 31 of Mahomed's paper on Chronic Bright's Disease without Albuminuria, Guy's Hospital Reports, 3d Series, vol. xxv.

[3] Transactions of the Association of American Physicians, vol. xii., 1897.

In the following table the clinical differences between the various forms of Bright's disease are set forth:

TABLE EXHIBITING THE CLINICAL DIFFERENCES BETWEEN THE PRINCIPAL FORMS OF BRIGHT'S DISEASE.

Acute Cases in which Dropsy occurs quickly and is extensive.

Acute Bright's disease; acute desquamative or tubal nephritis; acute parenchymatous nephritis; acute renal dropsy	Caused mostly by exposure, or scarlet fever. Dropsy extensive, generally begins in the eyelids or in the feet; usually fever; uræmia may be met with. Disease most common in childhood and among young adults. Recovery frequent; but disease may terminate in chronic parenchymatous nephritis.	Urine usually scanty, deep-colored, of high specific gravity, containing much albumin, often blood; also blood-casts; casts, many of large size, covered with epithelium, and a few hyaline and granular casts; and free epithelial cells, cloudy and granular; urea diminished.	Kidneys enlarged, congested or mottled, shedding epithelium; cortical substance increased; cones usually redder than cortical substance. Dilated convoluted tubes, distended with swollen, cloudy epithelium; at ends of tubules also blood or plugs of fibrin.

Chronic Cases in which Dropsy is variable in amount and may be absent.

Chronic parenchymatous nephritis; chronic tubal nephritis; chronic diffuse nephritis; large white kidney	History often of antecedent acute inflammatory attack; dropsy a prominent symptom. Marked anæmia; puffy face. Inflammations of serous membranes and uræmia not uncommon: hypertrophy of heart, especially of the left ventricle, or dilatation. Recovery possible, but doubtful.	Urine in normal or in increased quantity; specific gravity somewhat below normal; urea diminished; albumin generally in considerable amount; granular casts; at times compound granule-cells and partially fatty epithelium; no blood-casts; leucocytes.	Kidneys large, pale, capsules easily stripped off, cortical substance greatly increased; cones may be of natural color; tubes irregularly distended, and filled with granular epithelium and with detritus. Thickening of intertubular matrix.
Fatty Bright's kidney.	Persistent and obstinate dropsy, coming on gradually; face pale and puffed; hypertrophy of heart affecting often both sides. Always fatal.	Urine contains much albumin, fatty casts, fatty epithelial cells, free oil. Spec. grav. variable, usually from 1015 to 1030. Quantity variable, generally moderate or diminished; urea diminished.	Kidneys enlarged, and very fatty; sometimes have a mottled look. The tubes, especially the convoluted ones, full of highly fatty epithelium, and free oil.
Waxy kidney; lardaceous or amyloid degeneration of kidney	Follows usually wasting diseases, syphilis, caries, and long-continued suppuration. Rare in very early and in advanced age. Dropsy trifling, except late in disease; great emaciation; striking sallowness of face; liver and spleen enlarged; diarrhœa; much thirst; heart not affected; nervous symptoms infrequent. Unfavorable prognosis.	Urine increased, contains much albumin, but few casts, which are pale and transparent or highly refracting. The casts may or may not give the mahogany-red reaction with a watery solution of iodine. Spec. grav. low, yet usually above 1010; urea normal or slightly diminished.	Kidneys enlarged, smooth, and waxy-looking; capsule easily detached; cortex pale, anæmic; reddish-brown discoloration on testing with watery solution of iodine; cones often dark and congested. Morbid process at first chiefly along renal vessels.

TABLE EXHIBITING THE CLINICAL DIFFERENCES BETWEEN THE PRINCIPAL FORMS OF BRIGHT'S DISEASE.—*Continued.*

Chronic Cases in which Dropsy is variable in amount and may be absent.—Continued.

Chronic contraction of the kidney; contracted kidney; cirrhosis of the kidney; interstitial nephritis; granular kidney; fibroid kidney; gouty kidney..	Dropsy slight, frequently absent; face sallow; often headache and retention of urea, tendency to coma, and to convulsions; vertigo; anæmia; epistaxis; retinitis; hypertrophy of heart; liver may be cirrhosed. Most common between forty and sixty years of age. May exist for years unsuspected; is a very chronic disease.	Urine more copious than in health, yet extremely small amount of albumin, this at times temporarily absent; hyaline and large finely granular casts; altered epithelium; a little oil. Spec. grav. low; rarely above 1010, much oftener below; urea decreases gradually; marked decrease later in disease.	Kidneys waste slowly, become dense and contracted; capsule very adherent; surface often granular; thickness of the cortical substance diminished; cysts common. There is hypertrophy of connective tissue; compression and atrophy of gland-elements and of tubules. Cardio-vascular changes. Tissue changes in renal ganglia.

Diseases associated with Purulent Urine.

In every case in which pus in any quantity is detected in the urine, it becomes of great importance to ascertain primarily that it is not derived from the urethra, from the vagina, or from an abscess that has opened into the urinary passages. The first point we may decide by examining into the history of the case, and, if necessary, by an exploration of the parts, as well as by an examination of the urine procured in the manner recommended in the first part of this chapter; the second, by the same means, and by determining that a discharge takes place equally when no urine is voided; the third is more difficult to make out, but there is generally something in the symptoms and in the history of the case furnishing a clue to its interpretation,—such, for instance, as the sudden appearance of a large quantity of pus in the urine. Having excluded each of these morbid states as the source of the purulent urine, we next turn to see which of the maladies that are its most common cause is before us. They are:

Acute Cystitis.—Acute inflammation most frequently affects the mucous membrane at or near the neck of the bladder. It is much more commonly encountered in men than in women, and in adults than in children. Its main symptoms are a feeling of weight and pain in the hypogastric region, augmented by movement and by pressure. The pain does not, however, remain confined to the region about the bladder, but is felt also in the iliac and sacro-lumbar regions. It is attended with considerable febrile disturbance and extreme irritability of the affected viscus. The urine is voided drop by drop, and its passage is accompanied by straining and a scalding sensation at the

neck of the bladder; it is high-colored, cloudy from vesical mucus, and contains blood and pus and sometimes shreds of lymph. At first the urine is acid. The acute disease generally terminates within a week, leaving often an irritable bladder or a chronic inflammation.

The symptoms of acute cystitis are similar to those of *acute painful nephritis*, and the exciting causes may be much the same. But acute inflammation of the bladder differs from acute inflammation of the kidney by the far greater severity of the pain, its much lower position, and by the distress in voiding the urine. *Neuralgia*, or spasm, of the bladder may be distinguished from acute inflammation by the absence of fever, and by the sharp, lancinating, but paroxysmal pain, each onset of which lasts hardly longer than from two to six hours, and is attended with difficulty in passing water, which disappears as the pain subsides.

Metritis exhibits several of the traits of cystitis: we find the same hypogastric pain shooting to the thighs or to the anus and loins, the same feeling of weight in the perineum, and the same signs of irritation of the bladder and of fever. As it, however, generally occurs in the puerperal state, we have the history, and the character of the discharges from the vagina, to guide us, as well as the knowledge to be gained by a local examination.

Chronic Cystitis.—This affection, often called chronic vesical catarrh, is common in advanced age. It generally comes on in an insidious manner, and is excited by some obstacle to the evacuation of urine, such as a stricture, or by the presence of a stone in the bladder, or by an enlargement of the prostate gland. A paralysis of the viscus leading to retention of its contents, or a serious structural disease of its coats, whether malignant or non-malignant, may, however, also establish the morbid process.

The most usual symptoms, indeed in every way the most characteristic, are dull pain, a frequent desire to pass water, and the discharge of a large quantity of muco-pus or pus with each act of micturition. The urine, which is alkaline, on standing deposits a glairy, viscid sediment, in which, under the microscope, vesical epithelial triple phosphates, large pus-corpuscles, extremely regular both in contents and in shape, and pathogenic germs, especially the bacillus coli communis and the staphylococcus pyogenes, may be detected. The urine usually contains more albumin than is found in acute cystitis.

The diagnosis of the disease in males is easy. The only affection with which it is liable to be confounded is abscess of the kidney. In females, uterine disorders may so closely simulate it that it may require a local examination to tell the difference.

But, having decided the case to be one of chronic cystitis, it is always more difficult to discover its exciting cause. We have to depend, to a great extent, upon the history of the malady; its association with a stone can be determined only by the use of the sound.

Abscess of the Kidney.—This dangerous condition is the result of suppurative inflammation of the kidney, or of abscesses forming in connection with pyæmia, or with embolism. The suppurative inflammation is sometimes traceable to an acute attack of nephritis brought on by exposure or by external violence, to retention of urine, or to the impaction of a renal calculus; but at other times it originates without any assignable cause, and in an insidious way. The association of suppurative nephritis with erysipelas has engaged much attention, and the renal affection is even thought to be erysipelatous in its origin.[1] Abscess of the kidney may also arise from acute interstitial nephritis and in suppuration that occasions surgical kidney.

Abscess of the kidney is a rare disease. It has much the same symptoms as pyelitis. There is a fulness on one side of the spine associated with tenderness on deep pressure in the lumbar region, and with more or less constant pain, the pain and tenderness being increased by lying on the affected side; there are also fever and occasional rigors, digestive disturbances, and blood and pus in the scanty, acid urine, though pus in the urine may be absent. In some cases a marked tumor is found in the loin, extending towards the iliac fossa. If the abscess burst into the calyces, there occurs, simultaneously with a subsidence of the tumor, a sudden and copious discharge of pus with the urine, or, if it break into the intestine, with the fecal evacuation.

The disease almost never affects more than one kidney; hence so-called uræmic symptoms are rarely met with, since the healthy kidney enlarges and becomes capable of performing a double amount of work. Ebstein[2] has, however, observed that chronic abscess in one kidney may produce amyloid disease of the other. The disorder gradually leads in most cases to a fatal issue, from the irritation, the vomiting, the diarrhœa, the wasting discharge, and the protracted hectic; sometimes paralysis of one or both legs happens, adding greatly to the distress. There is a possibility of recovery, if the patient have strength enough to withstand the purulent drain until the abscess empties itself. It may do this through the urinary pas-

[1] Goodhart, Guy's Hospital Reports, 3d Series, vol. xix.

[2] Ziemssen's Cyclopædia.

sages, through the colon, through the lumbar muscles, through the diaphragm, and be evacuated by coughing, and the cavity of the abscess then cicatrizes; or the abscess may burst into the peritoneal cavity and cause rapid death.

The diseases for which the malady is most apt to be mistaken—leaving out those extremely rare cases in which abscesses from diseased vertebræ break suddenly into the urinary tract—are chronic cystitis, perinephritis, and pyelitis. From *cystitis* it may be distinguished by the dissimilar local signs and the different appearances of the urine. Thus, in the affection of the bladder the quantity of pus constantly discharged is far greater,—for in abscess of the kidney there are times when little or no pus is voided; on the other hand, the urine of the vesical disorder is less albuminous. In the renal malady we can detect casts and other renal products in the sediment.

Perinephritis unconnected with inflammation of the kidney is a very rare disease. When primary, it may result from exposure; but it is more generally due to contusion or strain. I saw an instance of it in a young man who, returning home from a long walk, strained his back in jumping a fence. An abscess gradually formed, giving rise to a slight fulness in the left lumbar region and severe pain, which disappeared as matter was discharged through the integuments. The function of the kidney was not affected.

But an external opening may be established when the process of inflammation and suppuration has begun in the kidney and thence spread to the loose tissues surrounding it. Under these circumstances, the appearance in the urine of pus prior to its discharge through the muscles of the back would be the only certain means by which we could judge where the suppuration had primarily taken place. The inflammation may travel upward from the pelvic viscera or from the head of the colon or the appendix; it has been also noticed after irritation of the testicles and of the spermatic cord. The pus is generally situated behind the kidney. Secondary perinephritis has been observed in pyæmia, and after typhoid and typhus fevers, smallpox, and the other exanthemata. The disease is not uncommon in childhood.[1]

The prominent symptom in perinephritis is pain, which at times is so severe as to confine the patient to bed with his knees flexed, with a sense of fulness and dragging weight, with tenderness in the region of the kidney, and with lameness owing to the interference with the play of the psoas muscles. The urine is generally unaltered,

[1] Gibney reports twenty-eight cases, Amer. Journ. of Obst., April, 1876.

or only full of urates; the bowels may be constipated, owing to the pressure of the tumor on the intestine. A rounded, doughy, and generally indolent swelling, uninfluenced by the respiratory movements, is usually found in the lumbar region or a little lower. The abscess may cause pulmonary or pleuritic complications, but rarely gives rise to jaundice. As the disease advances, severe chills, with high fever and copious night-sweats, occur, as well as emaciation and marked debility, and the thoracic symptoms may mask the renal; fluctuation may be at times detected, and, before the abscess breaks externally, a phlegmonous appearance of the skin where the abscess points is not unusual. Great relief follows the discharge of the pus.

From *inflammation of the psoas muscle* we distinguish perinephritis by the absence of marked sensitiveness over the renal region in the former complaint, and by flexion of the thigh in it producing pain.

Pyelitis.—Inflammation of the mucous membrane of the pelvis of the kidney is almost never idiopathic, being commonly caused by a calculus arrested in the ureter; or by a retention of urine from an obstacle in the ureter, bladder, or urethra; or by an extension upward from the bladder of an inflammation. Bright's disease and diabetes are not unusually, and typhus and the eruptive fevers, pyæmia, scurvy, diphtheria, carbuncle, puerperal septicæmia are occasionally, complicated with some degree of pyelitis. Pyelitis may be also catarrhal or rheumatic. Under these circumstances, and in all the infectious diseases, pyelitis is apt to show itself in an acute form.

The symptoms of the chronic malady are in part those produced by the morbid states exciting it, especially those denoting a calculus lodged in the kidney or arrested in its transit towards the bladder: partly those directly traceable to the inflammation of the pelvis and infundibula. The manifestations of the latter disorder are a constant dull pain in the loin, felt also in the course of the ureter, and the passage of pus and occasionally of small quantities of blood with the urine; in cases from retention and decomposition of urine there are recurring chills, sweats, vomiting, headache, delirium, and fever. In most cases of pyelitis the urine is acid, albuminous, very abundant, and offensive. It may be acid even if it abound in triple phosphates; if detained any length of time in the bladder it becomes ammoniacal. Bacteria are a frequent cause of pyelitis, as well as of abscess of the kidney, by migrating from a diseased bladder. In some instances of pyelitis an eruption like rubella is noticeable. Pyelitis not infrequently affects only one kidney.

The most difficult point connected with the recognition of pyelitis is the ascertaining that the purulent discharge does not proceed from

the bladder. And there is no positive sign to guide us, except the existence in the urine of epithelium from the pelvis of the kidney, distinguishable by its oval or fusiform shape, and by the frequent occurrence, in a cell, of clearly-defined, dark-colored, round granules, and of two nuclei. But this epithelium will not be always found, and we have then to fall back upon the history of the case, upon the attacks of renal pain, upon the hæmaturia caused by a calculus, and upon the combination of signs as pointing more to one disease than to the other. In some cases there is a perceptible swelling in the loin; at times, too, owing to coexisting degeneration of the cortex of the kidney, the amount of albumin is wholly disproportionate to that contained in pus, and this becomes a valuable indication of the affection not being vesical. But if there be a coincident disease of the bladder, the differential distinction may become impossible. Under these circumstances, too, the acid state of the urine, on which in uncomplicated cases much stress may be laid, is not apt to be a feature to aid us. The crystals of nitrate of urea formed when nitric acid is added to the urine have in pyelitis irregular blades or are in the shape of small feathers.[1]

Supposing the point settled, and the vesical origin of the pus disproved, the diagnosis is limited to an inflammation of the ureter, to an abscess in the substance of the kidney, and to pyelitis. Here again the history of the case comes into play. Furthermore, in the former of these affections—a very rare one, unless associated with pyelitis—the amount of pus in the urine is very trifling; in the second, too, it is less than in pyelitis, except when the abscess empties itself. The pus is also, as already indicated, not constant, alternately appearing in and disappearing from the urine; there is usually more obvious swelling, although this is by no means always discernible or even present in abscess, and the abscess is attended with much greater constitutional disturbance. Still, here again we must admit that the disorders are sometimes very obscure and difficult to distinguish, and it may be impossible to discriminate between them should the morbid states coexist, or a typhoid condition and uræmic fever be induced by the retention of the urine and its decomposition.

Catarrhal or rheumatic pyelitis is generally a short disease which ends favorably; so does the idiopathic pyelitis of the puerperal state, which rarely lasts more than from five to eight days. The pyelitis with retention and decomposition of urine is a much more serious complaint, and, although it usually runs a rapid course, not having

[1] Pascallucci, Il Morgagni, quoted in Lancet, June, 1873.

a duration of more than a week or two, it may become protracted. Pyelitis due to the *irritation of calculi* is apt to develop into a chronic condition.

In *tuberculous pyelitis* the symptoms are the same as in the ordinary form. The association with tuberculosis in other parts, and the detection of tubercle bacilli in the urine, establish the diagnosis.

In those cases of pyelitis in which there is a very decided obstruction to the flow of urine through the ureter, caused by a calculus, a clot of blood or viscid pus, or other débris, the discharge of pus is suddenly arrested and the cavity of the pelvis dilates greatly; gradually the gland-tissue is compressed, and a large pus-containing sac is formed, giving rise to a condition known as *pyonephrosis*, and to a distinctly limited swelling in the side. Tumors of this kind are ordinarily not painful to the touch, are indolent, and do not materially affect the general health, certainly not nearly so much as might be supposed. They frequently subside gradually by free discharges of pus, and the patient recovers.[1] Sometimes they become much reduced, and then swell up again from time to time. They may occur in both kidneys: but this is of great rarity. The urine generally contains albumin and considerable pus; it is acid and of low specific gravity.

Pyonephrosis cannot be distinguished from suppurative nephritis and ordinary *abscess of the kidney*, except it be by the history. The more constant and larger discharge of pus may be also made a point of diagnosis, as well as the obvious variations in the swelling, and the slighter constitutional symptoms. But too much stress must not be laid on these points; and the fact should not be overlooked that abscess of the kidney may be latent, or be present almost without fever, or with very obscure manifestations of pain, irregular attacks of fever, and vomiting, coming on at intervals for months or years.

When there is an impediment to the flow of urine the pelvis of the kidney dilates from the accumulating urine and we have *hydronephrosis;* in time the kidney tissue disappears. Hydronephrosis is due to mechanical obstruction from retroflexion or cancer of the womb, or from morbid growths or abscess of the bladder, or to congenital malformation of the ureter, or to movable kidney or to impacted stone in the ureter. Sometimes it is double; it is much more common in women than in men. The swelling to which it gives rise may subside simultaneously with a sudden and copious discharge of urine. When this symptom is absent, the diagnosis must be based on

[1] See, for instance, Cases XLVIII. and L. in Todd's Clinical Lectures on the Urinary Organs.

pain in the back, frequent micturition, and the existence of a fluctuating renal tumor, often lobulated, and on the absence of signs of suppuration. There may be attacks of renal colic due to the passage of clots of blood. The urine is at times copious, at times scant. The disease may lead to temporary, but entire, suppression of urine. Accurate percussion enables us to distinguish hydronephrosis from ascites; in the former the dulness is generally one-sided, and is uninfluenced by change of position. Ovarian cysts are more difficult to discriminate. Careful examinations by the rectum and by the vagina, and an investigation of the fluid after an exploratory puncture, are alone of value; and even the latter may mislead. Urinary constituents, for instance, have been found to be absent in rare cases of hydronephrosis. Pyonephrosis is chiefly distinguished by the irregular fever, chills, and the purulent urine.

Hydatid tumor of the kidney is of comparatively rare occurrence, and is likely to be confounded with hydronephrosis. When the urine contains no hydatid vesicles or their débris and the hydatid fremitus is absent, the diagnosis is extremely difficult, and must rest chiefly on the history of the case.

Ordinary *renal cysts*, when large enough to occasion a tumor, cannot be distinguished from hydronephrosis save by the history, and by the albuminous and decidedly bloody urine which the cysts give rise to, while in hydronephrosis the urine presents nothing peculiar, or occasionally only small amounts of pus and of blood. Then, renal cysts are double-sided, preserve the shape of the kidney, and do not rapidly change their size. There are casts in the urine, and the general symptoms are those of chronic interstitial nephritis including the cardio-vascular changes.

Pyelitis may be connected with fibrinous clots due to repeated hemorrhages from *multiple aneurisms of the renal artery*. We may suspect this condition if the other more usual causes of pyelitis seem to be absent, and if the affection happen in an old person having repeated attacks of hæmaturia and atheromatous arteries.[1]

Disorders in which a very large Amount of Urine is discharged.

Diabetes.—In diabetes mellitus, or glycosuria, the urine is of pale color, decidedly acid, and of high specific gravity, ranging generally from 1030 to 1050. The quantity passed is enormous: seventy pints and upward have been known to be discharged daily. The urea is

[1] Ollivier, Archives de Physiologie, 1873.

increased; so are the sulphates, the chlorides, and the earthy phosphates, while the alkaline phosphates vary greatly with the food, and uric acid is diminished; so is the coloring-matter. The urine contains from one to ten per cent. of sugar. In a small proportion of cases the flow of urine is not increased, nor is the specific gravity above normal. In some instances the phosphates are strikingly in excess.

The symptoms attending the drain of fluid from the system are great thirst, constipation, a dry, harsh skin, a red tongue, and a feeling of constant emptiness and of hunger. To these are added a steadily progressing waste of the body, muscular feebleness, chills, a somewhat hurried breathing, a peculiar mawkish odor of the breath, peevishness of temper, chronic catarrh of the stomach, a tendency to eczema and to boils and carbuncles, and in women pruritus of the vulva. The temperature is subnormal, often not over 96°. The knee-jerk is generally absent. Cataract and other defects of vision are not infrequent. There is a peculiar form of retinitis;[1] retinal hemorrhage and palsies of the muscles of the eyeball, diabetic hypermetropia, and atrophy of the optic nerves have also been noticed. Defects in accommodation are common. Diabetic endocarditis also happens, and is more frequent in women than in men;[2] and arteriosclerosis, neurites and neuralgias, periostitis,[3] and arthritic disorders[4] may have their origin in diabetes. Double sciatica is often of diabetic source: and there are cases presenting symptoms like those of tabes, with lightning pains and loss of knee-jerk.

Diabetes is generally a fatal disease; yet it is impossible to foretell its exact mode of termination. Some are cut off rather suddenly: others drag out a long existence, and die worn out and dropsical, or of cirrhosis of the liver, or of chronic nephritis, or of broncho-pneumonia, or of phthisis. For some days, or even for weeks, before death, the sugar may disappear from the urine.[5] Diabetic gangrene is also a mode, though not a frequent one, of termination of the disease.[6]

When the disease ends suddenly, it is apt to do so by so-called diabetic coma. The comatose condition is prone to be preceded by

[1] Galezowski, Compte-Rendu du Congrès Ophth. de Paris, 1862.

[2] Lecorché, Arch. Gén. de Méd., June, 1882; Bulletin de l'Acad. de Méd., 1880.

[3] Arch. Gén. de Méd., Feb. 1882, and Amer. Journ. Med. Sci., April, 1882.

[4] Dyce Duckworth, St. Barth. Hosp. Rep., vol. xviii., 1882.

[5] In a case for a long time under my charge, in which the diabetes lasted for several years, sugar entirely disappeared from the urine as the signs of phthisis became fully developed, for some months before death.

[6] See cases collected by Hunt, Transact. Phila. Co. Med. Soc., Nov. 1888.

vomiting and abdominal pain, rapid pulse, great anxiety and restlessness, labored breathing, depressed body-heat, headache, and drowsiness. These symptoms are attributed to the poisoning of the body by the development of *acetone*, a derivative of acetic acid, in the blood; the acetone can be found in the urine, and may be readily detected on the breath by its odor resembling that of chloroform. The evidence, however, of the decomposition of the sugar into acetone, and of the consequent nervous symptoms called diabetic coma, is not conclusive. Diacetone was believed by some to be a more probable cause; but betabutyric acid, from which acetone is derived, is now more generally thought to be the cause of the diabetic coma. Certain it is that this is due to some toxic agent of extreme acidity in the blood. In thirty cases of diabetic coma examined by Naunyn,[1] extreme acidity was found; and in twenty-six in which the examination was made, the proof of the excretion of large amounts of oxybutyric acid was conclusive.

Diabetes is a disease chiefly of the upper classes of society. It is very rare in the colored race, very common among Hebrews. It is especially found in neurotics who lead a sedentary life, and a connection between gout and diabetes can be often traced, as also between obesity and diabetes. The disease is vastly more frequent in men than in women, and is often hereditary. Mental emotion, worry, and excessive devotion to business are among its causes. There is evidence of its being contagious. The sugar is derived from the glycogen in the body, and when this forms in excessive quantities and is not fully destroyed in the lungs, it is excreted by the kidneys. But as the sugar-forming function is not a simple one, and various organs and structures, such as liver, pancreas, and nervous system, take part in it, and there may be even direct change of the food products into glycogen, the question of the origin of diabetes in a given case is never an easy one. Clinically speaking, we are apt to find diabetes in this connection: in large feeders, especially large eaters of the carbohydrates, with poor assimilative powers; the diabetics among the obese and the dietetic diabetics mostly belong to this group; in diseases of the liver, especially in cirrhosis, and there is a form of cirrhosis with enlargement of the organs and with pigmentation of the skin which is regarded as peculiarly associated with diabetes; in diseases of the nervous system, such as tumors, epilepsy,—in fact, in most various structural as well as functional disorders of the brain or spinal cord; in disease of the fourth ventricle, or of tumors pressing

[1] Diabetes Mellitus, p. 297, Vienna, 1898.

there, diabetes has been particularly noted; in diseases of the pancreas. The frequent association of pancreatic disease or disorder of its function with diabetes is very evident, and depends upon the withdrawal of the glycolytic ferment which the normal gland furnishes.

In the diagnosis of diabetes the constancy of the excretion of the grape-sugar must be regarded, and not merely its occasional presence. In mild cases the amount of sugar does not exceed two per cent.; in severe cases we find from five to ten per cent. In some instances the constitutional symptoms are very marked, and the disease runs an acute course. The sure test for diabetes is furnished by the chemical tests for grape-sugar in the urine, which have been discussed in an early part of this chapter. But blood-tests are also made use of, and are of value where sugar exists in doubtful traces, or where it is temporarily absent from the urine. Bremer's[1] test consists in comparing with each other slides smeared with normal blood and with the suspected diabetic blood, after having been heated in a thermostat to about 135° C., and cooled and stained in a one per cent. aqueous solution of Congo-red for two minutes. The excess of stain is washed off, and diabetic blood is found to be unstained or orange-stained, while normal blood shows the distinct Congo-red stain. In leukæmic blood, however, we may have the same result as in diabetic blood. Diabetic blood will turn weak alkaline solutions of methylene-blue to yellowish green or yellow, and Williamson[2] has, in accordance, suggested a blood-test for diabetes of definite proportion,—about a six per cent. solution.

Starchy and saccharine substances increase the quantity of diabetic sugar. Nay, they may be the cause of a little sugar appearing in the urine of healthy persons. Yet those in whom a saccharine state of the urine is readily induced are in danger of becoming diabetic. If we are in doubt whether we are dealing with a case of diabetes, we may follow Seegen's advice and let the patient eat heartily of saccharine and sugar-forming substances, and examine the urine three hours after the meal; if no sugar then be found in the urine, diabetes may be excluded.

In the aged, sugar may be present in the urine without being attended with distressing symptoms. It is in such cases that we are most apt to meet with the intermitting diabetes to which attention has been called by Bence Jones.[3] When the abnormal ingredient thus

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[1] Medical Record, Oct. 1897.

[2] British Medical Journal, 1896, vol. ii.

[3] Medico-Chirurgical Transactions, vol. xxxviii.

disappears from the urine, it is replaced by uric acid and by oxalates. There is still another form of intermitting glycosuria. Sugar is found in the urine during the paroxysms of intermittent fever; but it vanishes during the intervals.

Sugar is also found in the urine in small quantities in the obese, or after inhaling chloroform or taking chloral or sulphonal. Among the insane, sugar may be present in the urine without there being other symptoms of diabetes, and without grave significance.[1] Indeed, this appearance of sugar in the urine from passing causes or without other marked symptoms has given rise to the distinction made by some between *glycosuria* and *diabetes*, restricting the latter term to persistent saccharine urine with decided symptoms. The temporary glycosuria gets well; true diabetes rarely does.

In some instances we have *diabetes with coexisting albuminuria*, and even with other evidences of Bright's disease. In the majority of such instances the degeneration of the kidneys has happened subsequently to the diabetes, and in its more advanced stages, from their constant irritation; but I have met with cases in which the nephritis has preceded the diabetes. A high degree of fatty kidney or amyloid kidney has also been noticed in connection with diabetes. A small amount of albumin in diabetic urine is common.

Chronic Diuresis.—This disease is otherwise known as *polyuria*, or *diabetes insipidus*. It is characterized by the habitual discharge of a very large quantity of urine of low specific gravity, from 1001 to 1008, containing an excess of water, but no sugar; urea is increased; uric acid is very deficient; inosite is often present; kreatinin may be excreted in increased quantity. The general symptoms are much the same as those of diabetes; the thirst is generally extreme, and it may happen that more water is passed than is drunk. Most cases recover under treatment, except when dependent upon irremediable lesion. They sometimes die of suppression of urine.[2]

The cause of this singular malady is obscure. We meet with polyuria after cerebro-spinal fever, or in connection with tumors of the brain, or with disease of the medulla oblongata, or of part of the floor of the fourth ventricle, or with tumors compressing the abdominal ganglia. Lancereaux tells us that the disorder is not uncommon in syphilitic affections of the nervous centres;[3] and Bartholow's experience is that syphiloma of the brain is its most usual cause. I have

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[1] Lailler, quoted in Journal of Mental Science, May, 1871.

[2] Case under my charge at the Philadelphia Hospital.

[3] Sydenham Society's Translation, p. 77.

repeatedly encountered the malady after injuries to the head,[1] after sunstroke, or in persons broken down with malaria. At times it is seen in instances simply of great nervous depression without organic disease. It is, indeed, mostly connected with some abnormal state of the nervous system. It has been stated to coexist with marked excess of phosphates, and to be a phosphaturia.

Cases of chronic polyuria differ from true *diabetes* by the low specific gravity of the urine, and the utter absence of a saccharine ingredient. Sometimes a state of diuresis is found to exist temporarily during the removal of dropsical effusions, or when the action of the skin is insufficient. We also meet with apparent cases of diuresis in *hysterical women* and in persons who suffer from *incontinence of urine*. In all such we can establish the diagnosis by measuring the amount of urine passed in the twenty-four hours,—which amount may be large, but is not inordinate. In hysteria it may be temporarily very large after a paroxysm, but is not persistently so. In some instances diabetes mellitus alternates with diabetes insipidus. The discovery of an hydræmic centre in the cerebellum, as well as the well-known points at the floor of the fourth ventricle, which, according to the exact seat of puncture, produce increased flow of urine with sugar or without sugar, gives us the clue in which direction to look for the explanation of such cases. The large flow of urine we sometimes meet with in *contracted kidney* is known from hydruria by the presence of albumin and tube-casts and the other signs of kidney degeneration. An excessive flow of urine may happen in *hydronephrosis*. But the antecedent history, the previous existence, as a rule, of a fluctuating tumor, and the character of the urine, either normal or containing at times traces of albumin or of blood, will throw light on the character of the malady.

Disorders in which little or no Urine is Discharged.

Suppression of Urine.—Suppression of urine, unconnected with degeneration of the kidney, is a rare disorder. Yet it may occur in previously healthy persons, or in the course of fevers of low type, or in alcoholism, and probably associated with no other morbid state than congestion of the kidneys. It is occasionally met with as one of the freaks of hysteria, or is caused seemingly by the irritation reflected to a healthy kidney from a diseased bladder.

The symptoms it occasions, independently of the absence of the discharge of urine, are drowsiness, nausea, vomiting, coma, sometimes

[1] Transactions of the College of Physicians of Philadelphia, 1875.

convulsions; in one word, the symptoms of uræmic poisoning. The formidable complaint may give rise to marked urinous smell of the perspiration and of the breath, and to exceeding and very general cutaneous hyperæsthesia.[1] The temperature may be low, and remain so even if there be coexisting internal inflammation, or be above the norm.[2]

Concerning the exact cause of the supression we are often kept in the dark until the termination of the malady; for, unless familiar with the antecedent symptoms, we are unable to determine, in the absence of the urinary secretion, whether or not a disease of the kidney lie at the origin of the mischief.

Oppolzer tells us that we may diagnosticate *thrombosis of the renal vein* if we have diminution of the secretion of urine and its final suppression preceded by blood, albumin, and casts in the urine. If there be a history of severe injury to the kidney, these symptoms have a much more positive meaning.

Retention of Urine.—The urine retained in the bladder distends the viscus and forms a swelling in the hypogastrium, discoverable both by palpation and by percussion. The urine is generally not wholly kept back, for a slight discharge every now and then takes place, or there is a constant dribbling,—a matter which in itself should suggest the introduction of a catheter.

Retention of urine, if soon recognized, is not a dangerous complaint, as it can be at once relieved by the passage of a catheter; but if the ailment escape observation, or be inefficiently dealt with, the bladder may burst,—though Sir Henry Thompson tells us that this is a circumstance of exceeding rarity,—or the patient die from the absorption of the noxious urinary ingredients.

The causes which lead to retention are various; prominent among them, at least in a medical point of view, is paralysis of the bladder, especially that form of paralysis which occurs in low fevers; retention is also one of the symptoms of paraplegia; then inflammatory swelling of the neck of the bladder, organic stricture, or enlarged prostate may give rise to it; again, retention or incontinence may be due to

[1] This was the most obvious symptom in a case under my care at the Philadelphia Hospital, in which no urine was secreted for many days, the catheter being repeatedly introduced into the bladder. The patient recovered. She had, previously and subsequently to the attack, vesical catarrh. In a case reported by Fuller, St. George's Hospital Reports, vol. v., the difficulty existed for eight days without occasioning convulsions. It was the same in a case of mine that lasted eleven days and got well.

[2] Bourneville, Gaz. Méd. de Paris, 1872.

hysteria. If the urine be long retained in the bladder, it becomes alkaline, and putrefactive changes occur, and fission fungi, especially the micrococcus ureæ, develop in great numbers in the ammoniacal urine.

The disorder is readily detected. It may be discriminated from suppression of urine by the existence of the hypogastric tumor, and by the introduction of a catheter,—a means which, in cases of doubt, ought never to be neglected. Sometimes the abdominal swelling is so great as to lead to the belief of the existence of dropsy; and the error is fostered by learning that the patient has been passing his water, and has a constant desire to discharge it, or by seeing that it dribbles from him.

The retention from paralysis is distinguished from that due to other causes, as obstruction, by observing that the catheter enters readily, and that the urine flows out in a continuous stream, increasing and lessening with the respiratory movements, but does not come out in jets.

CHAPTER VIII.

DROPSY.

A COLLECTION of watery fluid in the areolar tissue or in the serous cavities constitutes dropsy. Now, dropsy is but a symptom, and is associated with various disorders; yet, though but a symptom, it is one that comprises so often apparently the whole complaint, that it will be useful to investigate connectedly the clinical meaning of its typical forms.

Dropsy, according to its Seat and Extent.

Dropsies may be external, or be confined to internal parts. To the latter variety belong hydrothorax, hydrocephalus, and ascites. External dropsies are illustrated by anasarca and œdema; the first, a universal accumulation of serous fluid in the areolar textures; the second, a localized collection in the same structures. Both exhibit painless swelling of the surface, devoid of redness; a skin often stretched and shining, pitting upon pressure, and retaining for some time the mark of the finger; and in both, the tumid part, if punctured, discharges a watery fluid. Œdema is most commonly perceived around the ankles; the tumefaction of anasarca is found generally not only in the lower extremities, but also in the arms and in the face.

Anasarca is usually dependent upon disease of the kidneys, or of the heart. The swelling rarely shows itself at all parts of the body at once; it ordinarily begins at the feet and ankles in diseases of the heart, in the face in diseases of the kidney.

Œdema may be due to the same causes. Yet a limited collection of fluid is often the consequence of a purely local difficulty, of a character interfering with the venous circulation. Thus, the compression or obliteration of a large vein occasions œdema below the point of the disorder. We see œdema happening if swollen glands press upon the main vein of a limb. We also meet with it in the adhesive form of venous inflammation, and in phlegmasia alba dolens. In all of these forms the œdema is one-sided, and there is little difficulty in its recognition. A circumscribed œdema also accompanies erysipelatous inflammations of the skin or subjacent tissues, and is found in limbs the general nutrition of which has been lowered by paralysis.

When the external dropsical effusion is dependent upon a tumor seated in an internal cavity and interfering with the passage of the blood, it may be very local and one-sided, as we sometimes find in connection with abdominal cancer; but it is most apt to be found on both sides of a portion of the body, although more particularly marked on one side. The œdematous extremities exhibit usually also marked enlargement of the veins.

Another source of a double-sided œdema is anæmia. The serum collects first about the ankles. The absence of any discoverable organic affection, the pallid countenance, and the pearly whiteness of the conjunctiva are very significant. A microscopical examination of the blood and a blood-count establish the diagnosis.

A dropsical effusion in part of similar origin, but much more often connected with *internal dropsy*, especially with ascites, is the dropsy we observe in those broken down by malarial poisoning. The state of the liver and spleen, or of the kidneys, added to the condition of the blood, determines the greater extent of the effusion.

Dropsy, according to its Causation.

Having viewed anasarca and œdema as in the main uncombined with internal dropsies, and as forming the sole signs of the dropsical complaint, let us now look at them when associated with effusions of serum elsewhere. The same remarks will also apply to hydrothorax and to ascites, the meaning of which, when occurring alone, we have inquired into, but which we shall here consider in their relations to *general* dropsy, or that form in which anasarca or œdema coexists with dropsy of one or several of the large serous cavities.

First, let us examine into the causes of general dropsy. The most common are a disease of the heart, of the kidneys, or of the liver; so common, in truth, that in every case of dropsy we must always examine these organs carefully. According as the dropsical accumulation originates in a morbid state of these viscera, it is called cardiac, or renal, or hepatic.

Cardiac dropsy arises in consequence of the deranged or enfeebled circulation produced by a disease of the walls and cavities of the heart, associated or not with a valvular lesion. The dropsy begins in the feet and ankles, being much influenced by position, and gradually extends upward; but it is rarely very obvious in the face or upper extremities. The thighs and scrotum are sometimes greatly swollen, and there is a watery effusion into the pleural cavities or into the pulmonary parenchyma. Cardiac dropsy is generally chronic.

Renal dropsy is usually much more general than cardiac dropsy.

It is often first noticed in the face and eyelids. The proof that the dropsy is renal is furnished by the presence of albumin and of casts in the urine. Renal dropsy is very often acute, attended with active symptoms, and occurs in the course of acute nephritis. The history frequently points to exposure to cold and wet.

Occasionally the dropsy is owing to an affection both of the kidney and of the heart; and the inquiry may arise, which of the organs was primarily disturbed and gave rise to the dropsy? Valve-disease makes the cardiac, simple enlargement of the heart with decided amounts of albumin and a large number of casts especially of granular character makes the renal, view predominant.

Hepatic dropsy may, like the preceding forms, be more or less general; but it is rarely so, unless of long standing, or unless there be coexisting disease of the heart or of the kidneys. The most usual kind of dropsy of liver origin is abdominal dropsy; indeed, ascites is frequently looked upon as constituting a proof of hepatic disorder. Ascites may be also produced by peritoneal tumors or inflammation, by enlargement of the spleen or of the pancreas, or by the pressure of diseased glands,—in fact, by any lesion which occasions a decided impediment to the portal circulation.

Again, it is possible, though it is not often a cause, that mere irritation of the *areolar tissue* will occasion more or less general dropsy. This was a favorite doctrine of the older physicians; and H. C. Wood thus explains the dropsy of arsenical poisoning.[1] Another cause of general dropsy, especially of anasarca, is *peripheral multiple neuritis*. I have seen this in cases in which the electric reactions, the absence of the knee-jerk, the altered sensation, made the diagnosis clear.[2]

Besides these sources of general dropsy, we may find deterioration of the blood, with, perhaps, a simply enfeebled condition of the heart, giving rise to it. But such a state is much more likely to occasion œdema, or anasarca, than general dropsical effusions.

There is a disease apparently like anasarca, but unlike in the absence of serous fluid in the connective tissues. It is the disease pointed out by Sir William Gull as a cretinoid state, and called by Ord *myxœdema*, consisting in the progressive invasion of the connective tissues of the body by a mucin-yielding substance, unassociated with albuminuria or disease of the heart, but invariably combined with destructive change and decrease of the thyroid gland. It affects chiefly adult women, who present swollen, waxy-looking features,

[1] Amer. Journ. Med. Sci., July, 1871.

[2] As in a case seen with Dr. Lewis Brinton.

with not infrequently a circumscribed flush on the cheeks, and who are markedly anæmic, and seem to have an excess of subcutaneous fat. The skin is everywhere thickened and rough, is devoid of perspiration, and the puffy integuments do not pit, or pit but very slightly, on pressure. The eyelids are greatly thickened and hang in folds; the nose is broadened; the lips are swollen, as are the tissues above the clavicles. The hands are often swollen and misshaped, the nails are brittle; there is loss of teeth and of the hair; the thyroid gland can generally not be felt. The temperature is below the normal; the excretion of urea is diminished. The expression is dull and heavy. The movements of the limbs are slow and languid; the gait is uncertain and awkward; sensation is impaired; there is irritability and suspiciousness of temper, with increasing hebetude, monotonous voice, slow, drawling speech; finally, melancholia and aberration of mind may supervene. The disease may be artificially produced by the removal of the thyroid gland. It has been also noticed after the long administration of iodide of potassium.[1] In certain cases the atrophy of the thyroid is preceded by hypertrophy.[2] The disease may be preceded or be attended with intractable uterine hemorrhage without apparent cause;[3] bleeding from the nose and gums also occurs.

The swelling of myxœdema is distinguished from the dropsy of *acute nephritis* by affecting the forehead as well as the face, by the mental symptoms, by the absence of decided pitting, by thickening of the alæ of the nose, and by the results of the urine examination.

It is more difficult to distinguish *contracted kidney* from myxœdema. In both we may find excessive flow of urine of low specific gravity, and a few hyaline casts associated with very small amounts of albumin. But if we have dropsy in contracted kidney, it is in the lower extremities. Then, the skin is not dry and desquamating, and there is not the physiognomy nor the state of mind of myxœdema.

Adiposa dolorosa,[4] a disease described by Dercum, differs from myxœdema in the irregular and painful fatty masses not being found on the face, hands, and feet, and in the absence of mental and psychical phenomena.

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[1] Stalker, Lancet, Jan. 1891.

[2] Ord, quoted in Sajous's Annual, vol. iv., 1891.

[3] Kirk, Lancet, Sept. 1893.

[4] University Med. Mag., Dec. 1888, and Twentieth Century Practice, vol. xi. See also Eshner, Phila. Med. Journ., Oct. 1898; Spiller, Med. News, Feb. 1898.

CHAPTER IX.

DISEASES OF THE BLOOD-VESSELS.

Only a short description of these will be here given, since many have been already mentioned in connection with other maladies, and our knowledge of others is still pathological rather than clinical.

Diseases of the Arteries.

The principal of these are inflammation and atheromatous changes.

Arteritis.—Inflammation may attack the outer coat, *periarteritis*, the inner coat, *endarteritis*, or all the coats, *general arteritis*. All these processes may be the result of rheumatism, of gout, of syphilis, of lead poisoning, of infective maladies, or of inflammation spreading from surrounding textures.

In *periarteritis* the last-named is the most common cause. The large arteries are the ones that are pre-eminently affected, and inflammation of the external coat of the thoracic aorta is more often encountered than that of any other artery. It may be acute; occasionally it has its origin in inflammation of the inner coat. It may lead to suppuration, and, the pus finding its way into the caliber of the vessels, pyæmia and metastatic abscesses are caused. But it is not possible to make a certain diagnosis of the condition.

There is a peculiar disease of the arteries, *periarteritis nodosa*, which, with the signs of acute desquamative nephritis and fever and marked anæmia, gives rise to numbness, to rapid loss of muscular power with deficient electro-muscular contractility, and to such severe muscular pains that they are really mistaken for those of trichiniasis. But the history of the ailment, the signs of the thickening of the vessels, the little nodules under the skin, if discernible, the violent paroxysmal pains in the hypochondrium, the spreading paralysis, starting from the fingers as the malady advances, and the rapid pulse with the comparatively low temperature, throw light on the cause of the muscular distress. The disease is rapidly fatal.

Endarteritis is almost always chronic, and chronic endarteritis is most commonly due to rheumatism, to gout, to syphilis, to alcohol, to the poisonous influence of lead or of arsenic, to altered quality of the blood, or is seen in connection with contracted kidney. As regards

the latter, the question may arise as to whether it has caused the change in the arteries or is a mere coexisting affection owing to the same general morbid process, a fibrosis. Arthur V. Meigs[1] urges this view, and I believe it is generally the true explanation. It is certain that chronic endarteritis is found without Bright's disease, or preceding it, and gives rise to symptoms by which it can usually be recognized. It is commonly described as *arteriosclerosis*, in consequence of the hardening of the walls of the artery, and its most usual kind, the senile form, is noted after the age of fifty as a degenerative change. Thickening of the intima is the most common disease of arteries, and may lead to obliterative endarteritis.[2]

The thickening of the intima of the arteries and arterioles may extend to some degree into the veins. The symptoms to which chronic endarteritis gives rise are increased blood-pressure, headache, cold extremities, breathlessness on exertion, anæmia, epistaxis, or hemorrhages into internal organs, such as the brain or the lungs; œdema without recognizable cause; attacks of bronchitis or catarrhal pneumonia; and torpor of the liver. An appearance of prominence of the smaller vessels and their greater resistance show the fully developed disease, and we then find nervous symptoms, such as vertigo, at times with syncope, loss of memory, and general want of power in the limbs. Hypertrophy of the heart, fibroid heart, and, at times, dilatation and valve-changes may also be present, as well as albumin and casts in the urine, and other signs of kidney affection. But these do not necessarily occur. Again, there are cases in which they seem to precede the endarteritis. The visceral complications of the malady make statements about the temperature uncertain, but I believe that it is persistently slightly elevated. Endarteritis is at times compensatory in slowing of the blood-current.[3] An accentuation of the second sound, as well as its reduplication, is a usual feature in arteriosclerosis. In arteriosclerosis of the coronary arteries the pulse is slow or irregular, and angina pectoris is common.

Extensive inflammation of the arteries, a *general arteritis*, is a very rare affection, and when it happens it is acute. In a few instances of rheumatism we find new vessels arising, and especially inflammation of the fibrous structures of the aorta. This condition may be suspected should we observe intense general uneasiness and distress.

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[1] Transactions College of Physicians of Philadelphia, 1888 and 1889, and the Organs of Disease, Philadelphia, 1897.

[2] The changes by which this is brought about are admirably shown in Meigs's work on the Organs of De

[3] Thomas Yeoman's , 1888.

with pain, increased pulsation, a distinct murmur in the course of the vessel, and tumultuous action of the heart without there being obvious signs of disease of that organ present. Still, the diagnosis is never a positive one. We may also meet with arteritis clearly infective, and general or local, in influenza, in pneumonia, in typhoid fever, and in ulcerative endocarditis. The result of the inflammation is that the blood may clot, and thrombi or emboli result, and, if infected, pyæmic fever develop. It is generally impossible to recognize the malady until after the thrombosis; and then severe pain in the limb supplied by the affected vessel, its sensitiveness and cord-like feel, the absent pulse and the coldness of the skin and lowered local temperature, and the swelling of the part are significant of a condition that often ends in gangrene. Yet all these signs of narrowing of the caliber of a vessel may occur without a thrombus, and be due to proliferating endarteritis, such as may exist in *obliterative endarteritis.*

Atheromatous Changes.—These are only the more obvious naked eye appearances, especially as they are found in the aorta and larger vessels, due to arteriosclerosis; calcareous degeneration is often seen. These alterations, happening in internal arteries, are beyond the accurate discernment of the physician. He may infer that they exist, if a distinct systolic blowing sound be heard in the track of the aorta or its branches, in a person who is not markedly anæmic, who is past middle life,—and therefore at an age at which these kinds of changes of tissue happen,—or has had any of the diseases predisposing to arteriosclerosis, and in whom no cardiac murmurs, or only faint cardiac murmurs, are perceived. But it is chiefly by the age of the patient, the rigid resisting superficial arteries, often irregular to the touch, and the gradual development of cardiac enlargement, that a conclusion as to the meaning of the physical signs is arrived at. The atheromatous change may be so great as to cause almost complete occlusion, even in arteries as large as the common carotid.

Diseases of the Veins.

The chief affection of the veins in a diagnostic point of view is inflammation.

Phlebitis.—This is met with by the surgeon much oftener than by the physician, who encounters it more especially in affections of internal organs, such as the liver, and has to study it in association with the formation of thrombi, and metastatic abscesses to which it leads, and with infective fevers. The most common form in which phlebitis comes under the cognizance of the physician is in connection with milk leg, or *phlegmasia alba dolens.* Here we have

usually phlebitis with an obstruction by a coagulum of the venous circulation in the affected limb, and bacilli, those of typhoid fever for instance, have been detected both in the clots and in the walls of the vessels. Yet it is by no means certain that the thrombosis is always secondary and caused by phlebitis. The phlebitis or the thrombus that forms, when of septic origin, may lead to pyæmia. The disease, except in gouty phlebitis, is mostly one-sided. The pain in the leg may cause it to be mistaken for rheumatism, but the one-sided swelling and the œdema distinguish it. Among its early and significant symptoms is pain on pressing the calf of the leg on the affected side.

Diseases of the Capillaries.

Some of the organic diseases of the capillaries belong to the arteriosclerosis in Bright's disease, or to the waxy degeneration in purpura. It is difficult to say what the functional disorders are, for many of them are regarded as forming part of the peripheral diseases of the nervous system, and the affection of the arterioles and of the capillaries is a mere vasomotor spasm in connection with the neurosis. This is supposed to be the case in the anomalous localized sensations of cold which some patients have in particular parts of the body, though their persistency is unlike a spasm. The painful flushings of the feet bespeak temporary excessive dilatation of the fine vessels.

A spasm of the minute vessels of more permanent character may lead to profound disturbance of nutrition in a part, even to its destruction. This is the case in the vasomotor neurosis, called *symmetrical gangrene*, or "Raynaud's disease."

The affection shows itself in three forms, local syncope, local asphyxia, and symmetrical gangrene, which are in reality but different stages of a condition in which there is recurring contraction of the arterioles and consequent interference with nutrition. The malady is most often seen in the hands affecting corresponding fingers; it is also met with in the feet, on the exterior surface of the forearm, and sometimes in the helix of the ear, on the nates, the front of the thighs, and below the knees. It is nearly always symmetrical. The local syncope shows itself mainly in sudden attacks of pallor, coldness and numbness of corresponding fingers, and in these "dead fingers" there is a cramp-like pain and impairment of tactile sense and of sensibility to pain; the surface temperature is lowered. The attacks are apt to come on at the same hour, often in the morning, and may recur daily for some months. They are more common in winter than in summer, are readily brought about by exposure to cold or by putting the hands in cold water, and are especially met

with in hysterical women and in neurasthenics. Each attack lasts from a few minutes to several hours; in the reaction the skin becomes red and sensitive to pressure. In local asphyxia we have the same history, but duskiness is soon noted, and purple or bluish discoloration of the symmetrically affected parts. There is much pain in them, and difficulty in executing concerted movements. The paroxysm gradually passes away; at times there are coexisting temporary alterations in the fundus of the eye. In symmetrical gangrene there may have been preceding local syncope or asphyxia, but these have become very frequent, and the altered nutrition shows itself in bullæ forming, and then in limited gangrene, as of the tip of a finger, which slowly sloughs off; within ten days, generally, the gangrenous process is over.

The local character of the lesions, their intermittency, and their superficiality, are the chief features of Raynaud's disease. We do not find lesions of the vessels as in senile gangrene. The malady is closely allied to paroxysmal hæmoglobinuria, which, indeed, has been repeatedly observed in association.

Raynaud's disease must not be mistaken for *chilblains*. These do not appear, disappear, and reappear in the manner in which the discoloration does in Raynaud's disease.

In *erythromelalgia*, described by Weir Mitchell,[1] there are vascular changes, acute congestion, or cyanosis. The disorder manifests itself in one or more extremities, usually in the heel or the sole of the foot, and is attended with flushing, local fever, and great pain, which comes on in paroxysms, aggravated by the vertical position and by movement. It is an affection of middle life, of which the pathology is still undetermined. It resembles most closely Raynaud's disease. But contrasting *Weir Mitchell's disease* with this, we find these striking differences: there is in erythromelalgia no change of color until the part hangs down, when it becomes rose-red. Then, too, the pain becomes worse, as it also does in summer and by heat, whereas neither position nor season affects the local asphyxia of Raynaud's disease, though cold is very apt to produce it. Moreover, in this there is lowered local temperature and anæsthesia to touch and pain, whereas increased heat of the flushed part, undisturbed sensation, and hyperalgesia mostly occur in Weir Mitchell's disease. Further, this is generally symmetrical, and never associated with a local gangrene, such as often follows the local asphyxia of Raynaud's disease.

[1] Medical News, Aug. 1893.

CHAPTER X.

DISEASES OF THE BLOOD.

Prominent among the clinical traits of all diseases of the blood are general debility, a changed aspect of the mucous membranes and of the skin, especially in color, and alterations of nutrition. In the investigation of these diseases, the microscope is of the first importance. It informs us with regard to the relative proportions of the white and red corpuscles, and exhibits the blood-plates or hæmatoblasts. It tells us much as to what part of the blood-making organs the former are derived from, and which are purely pathological; it indicates whether the red globules are of the right color, whether their outline is regular, and whether their number is altered. It enables us to study the blood-films and the effects on them of various stains.

To count the blood-corpuscles, the forms of apparatus now mostly in use are the hæmocytometer of Thoma-Zeiss and of Gowers. Another is the graduated moist-chamber globule-counter of Malassez; another the hæmatokrite.

The Thoma-Zeiss, or Zeiss, hæmocytometer consists of three parts: a graduated pipette or mixing-vessel, with rubber tube attached; a counting-cell on an object-slide made of ground glass; a cover-glass with ground level surfaces.

To count the red corpuscles of the human blood, the tip of the finger should be thoroughly cleaned, the middle finger of the left hand being generally selected. By rubbing the end of the finger or the lobe of the ear with a coarse towel a slight hyperæmia is induced, so that a cut with a spear-pointed needle will permit of the flow of a drop of blood sufficiently large for examination. The tip of the pipette is placed into this drop, and the blood carefully drawn up to the mark 1,—*i.e.*, one cubic millimetre. After this has been accomplished, the tip should be cleaned by means of a soft cloth and the pipette inserted into a carefully filtered ten per cent. solution of sodium sulphate, or Thoma's substitute of a three per cent. solution of sodium chloride, or Gowers's solution of 112 grains of sulphate of sodium in 5 drachms of acetic acid and 4 ounces of water. This is drawn up

into the tube until the bulb is filled to the mark 101. The blood and fluid are then mixed by shaking the tube, holding the finger over the tip of the pipette, that the liquid may not escape. After the mixture has been thoroughly effected, half of the fluid in the bulb is blown out, and the drop that follows is permitted to flow on to the previously cleaned floor of the counting-cell. The cover-glass is then immediately placed in position, and the apparatus allowed to stand upon a horizontal surface for two or three minutes, that the corpuscles may settle. For the success of this operation perfect cleanliness must be maintained.

In order to make the examination, the slide should be placed in the stand of the microscope and held in a horizontal position, that the corpuscles may not be displaced. Great care should be taken that no liquid flow between the cover-glass and the ring. It is important that the drop of blood mixture shall remain standing in the centre of the cell, and that by the spreading of the cell the under surface of the cover-glass shall be in contact with the mixture for several millimetres. Using a one-fourth or a one-fifth objective glass to bring into view the divisions cut upon the floors of the cell, we find that upon these lie the red blood-corpuscles. The number of corpuscles in each space is then noted, counting in the corpuscles touching the top and right lines, but leaving out those touching the lower and left lines. Through each fifth horizontal and vertical row of the lines an additional line is drawn, for the purpose of fixing more readily the position of the squares counted.

Each field of the net-work contains a surface of one four-hundredth of a square millimetre. The distance of the cell-floor from the under surface of the cover-glass is one-tenth of a millimetre. Each square, therefore, represents the one four-thousandth of a cubic millimetre. The number of corpuscles contained in one of these cells multiplied by the number of times the blood has been diluted will give the amount of corpuscles contained in the one four-thousandth of a cubic millimetre. The amount contained in a cubic millimetre can, therefore, be found by multiplying by four thousand. The surest method is to count at least thirty-six spaces, as Cabot[1] does, or forty spaces, to take the average of them all, and proceed as above. It is sometimes difficult to distinguish the white from the red blood-corpuscles, and this difficulty is obviated by adding a one-third per cent. solution of acetic acid to the diluted blood. Another method for computing the white corpuscles and their relative number to the red is to

[1] Clinical Examination of the Blood, 1898.

use, with the salt solution, a few drops of a one per cent. solution of gentian violet; this leaves the red blood-corpuscles unaltered and stains the leucocytes a deep violet; or we may employ Toisson's solution, which consists of methyl violet, 5B, 0.25 gm.; chloride of sodium 1000 gms., sulphate of sodium 8000 gms., neutral glycerin 30,000 cms., and distilled water 160,000 cms. It takes about ten minutes to fully stain the leucocytes.

FIG. 68.

Potain's pipette.

The following method for differential counting of leucocytes in fresh blood is recommended by Elzholz. After drawing blood into the pipette, a solution composed of seven grammes of two per cent. eosin solution, forty-five grammes of glycerin, and fifty-five grammes of water is added; then, a solution composed of four drops of concentrated watery solution of gentian violet with one drop of absolute alcohol and fifteen grammes of water, by which the polynuclear cells are more deeply stained; the eosinophile cells are reddish violet.

The *hæmocytometer* of Gowers is about the same as that of Zeiss, differing mainly in the number of divisions on the cell, each space being but one-tenth of a millimetre in length. The method of preparing the blood solution is not so convenient as that of Zeiss. A hæmic unit of five millions of corpuscles to one cubic millimetre of blood is assumed. The pipette is best cleaned with an aspirator. In the hæmocytometer of Durham[1] the pipette is self-filling.

By the original method of Malassez the blood is diluted with artificial serum so that it represents $\frac{1}{100}$ or $\frac{1}{200}$ of the original. A small amount is then introduced into a flattened capillary tube of known capacity and, with the micrometer eye-piece, the globules are counted in the capillary tube of a certain length, say 500 micromillimetres. The capacity of this length of the tube in parts of a cubic millimetre being already known, the entire number of globules in a cubic millimetre of the undiluted blood is easily determined by calculation. For the purpose of diluting the blood[2] Potain's capillary pipette (Fig. 68) is well adapted.

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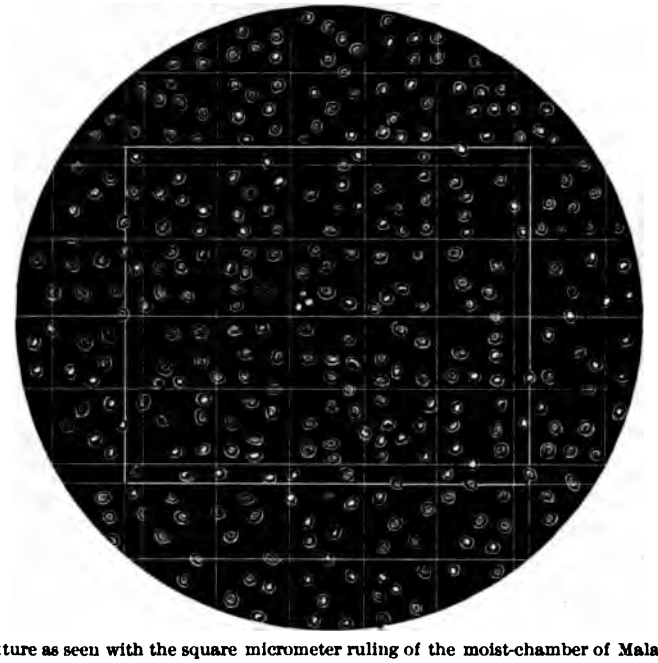
[1] Coles, Edinburgh Medical Journal, Oct. 1897.

[2] Malassez recommends for artificial serum a five or six per cent. solution of sodium sulphate, having a specific gravity of 1020 to 1024.

The use of the hæmocytometer in any form requires skill and patience, and even with great care the counts give between two and four per cent. of error. To save time and prevent the eye-strain, Hedin,[1] in 1890, devised the *hæmatokrite*, by which the entire mass of the globules in a definite quantity of blood can be rapidly ascertained. The

FIG. 69.



Blood-mixture as seen with the square micrometer ruling of the moist-chamber of Malassez; magnified 250 diameters.

instrument consists of a capillary glass tube, correctly graduated, in which a certain volume of diluted blood is held, while the tube is subjected to centrifugal action, by which the separation of the plasma and the cellular elements is effected. The proportion is determined by the scale engraved upon the side of the glass tube, and the globular richness of the blood is promptly determined. The original instrument of Hedin has been advantageously modified by Gärtner,[2] Arnold,[3] and especially Daland.[4]

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[1] Scandinavisches Archiv für Physiologie, No. 2, 134; Prager Med. Wochenschrift, 1891.

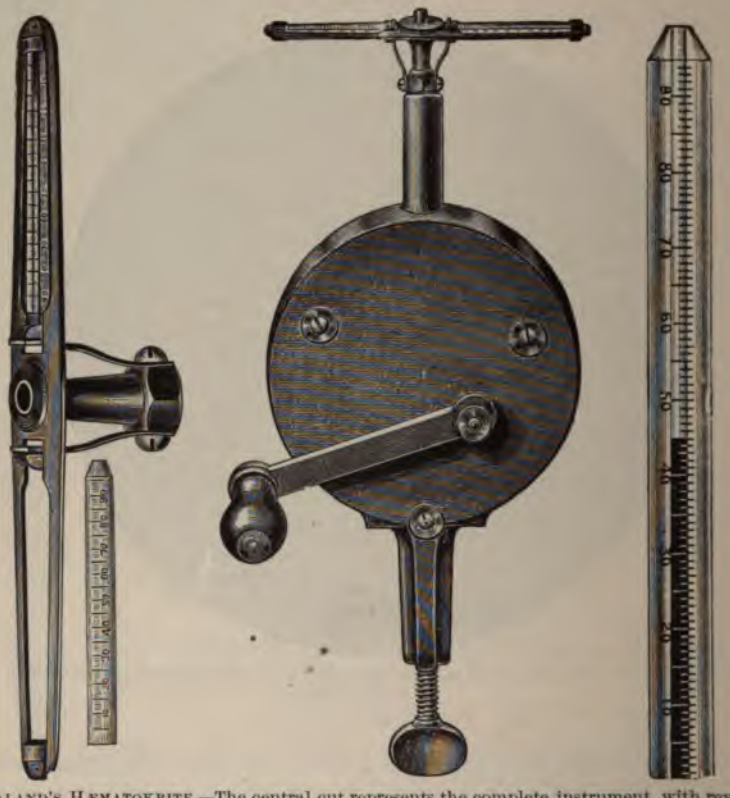
[2] Berliner klinische Wochenschrift, 1893, No. 4.

[3] Medical News, Sept. 29, 1894, p. 348.

[4] Transactions of the College of Physicians of Philadelphia, May 2, 1894.

The instrument of Daland is arranged to carry two glass tubes, the outer ends of which fit into small, cup-like depressions. Each tube measures fifty millimetres in length, with a lumen of half a millimetre, and upon it is a scale representing one hundred equal parts; a lens front, by magnifying the column of blood, facilitates the reading

FIG. 70.



DALAND'S HÆMATOKRITE.—The central cut represents the complete instrument, with revolving tubes in position. To the left is the tube-holder or frame, with one tube removed so as to show the spring, by which the tube is to be held in place. To the right is a tube containing blood that has been subjected to centrifugal force, indicating ninety per cent. of corpuscular elements, as compared with normal blood.

of the scale. A single revolution of the large handle causes one hundred and thirty-four revolutions of the frame. The instrument must be firmly secured to a solid table. The method of employing the hæmatokrite is simple. To fill the glass tube, a rubber tube is slipped over the end of the capillary pipette, and to the extremity of this rubber tube a mouth-piece is attached, precisely in the same manner as when the hæmocytometer is used. The glass tube or pipette must be absolutely clean and dry. The finger of the patient is punctured; the

blunt point of the pipette is to be placed into the blood, and the tube completely filled by suction. The finger of the operator is then quickly applied to the blunt extremity of the tube, which is next inserted into the frame, and rotated at the rate of ten thousand times per minute. All that remains is to read the percentage volume of blood from the scale. The divisions on the pipette are one-half millimetre apart, so that the scale can be read without difficulty.

The entire procedure need not occupy more than three minutes. In health the volume of red corpuscles is a little over fifty per cent., so that by doubling the number as read from the scale we can get a proportionate expression of the percentage of corpuscles in a specimen as compared with the normal. With the hæmatokrite of Daland, when a column of red corpuscles obtained from a healthy man is examined, the white cells present a sharp, clearly defined, and shallow white band. When the leucocytes are much diminished in number, this white band is imperfect, and in places the red color of the biconcave disks is visible. In the Arnold-Hedin hæmatokrite, diluted blood is used, and the readings are made more accurate by the use of a low-power microscope; the motor runs by electricity. Cabot objects to the hæmatokrite on account of the noise it makes.

Exact results cannot be obtained by any method, and, as shown by Henry, there is a diurnal variation in the number of the corpuscles in health. Normal blood contains about five million red blood-corpuscles, nearly ten thousand white blood-corpuscles, and two hundred and fifty thousand blood-plaques or hæmatoblasts,[1] to the cubic millimeter, and each red corpuscle holds in suspension a certain percentage of hæmoglobin. Any marked variation in the number of corpuscles, or the relation of red to white, or in the amount of hæmoglobin, is indicative of an abnormal state. For accuracy it is always requisite to study the relationship between the different elements of the blood.

In estimating the number of red blood-corpuscles age and sex must be taken into account. In healthy women the number per cubic millimetre is somewhat less than in healthy men, being about four million five hundred thousand; in new-born infants it often exceeds six million, as both Hayem[2] and Henry[3] have found by repeated observations. But in the infant the constitution of the blood is remarkable for its variability. The very suggestive observations of John

[1] Hayem, Du Sang, Paris, 1889.

[2] Du Sang et de ses Altérations anatomiques, Paris, 1889.

[3] Amer. Journ. Med. Sci., April, 1890.

K. Mitchell[1] have shown that in adults massage increases enormously for the time being the number of red corpuscles in the blood count. Prolonged fatigue diminishes them, so does pregnancy.

The white blood-corpuscles in normal healthy blood are in the proportion of about one to six hundred of the red, this varying somewhat in different individuals without being indicative of disease. When the red blood-corpuscles are reduced in number, the proportion of leucocytes is greater, without there being necessarily an increase in the number. The safest method of procedure is to estimate the number of white corpuscles to the cubic millimetre, so that any increase or diminution in their amount will give their true condition irrespective of the change in the number of red disks.

FIG. 71.

The hæmoglobinometer of Gowers. A, bottle with pipette-stopper; B, capillary pipette; C, graduated tube; D, tube containing standard tint, fixed in E, a wooden block; F, guarded needle.

The chief apparatuses for *estimating the hæmoglobin* are the hæmoglobinometer of Gowers, Fleischl's hæmometer, Hénocque's hæmatoscope, and Oliver's hæmoglobinometer. Of these, the hæmometer of Fleischl is the most used. Hénocque's is especially valuable for spectroscopic examination. In Oliver's hæmoglobinometer the blood tint is compared with definite tints of glass. Gowers's apparatus consists of two glass tubes of exactly the same size. One contains a standard of the tint, of the dilution of twenty cubic millimetres of blood with one thousand nine hundred and eighty cubic millimetres of water. The second tube is graduated to one hundred degrees,

[1] Transactions of the College of Physicians of Philadelphia, 1893.

which equal two cubic centimetres. The twenty cubic millimetres of blood are measured by a capillary pipette. This quantity of the blood to be tested is dropped to the bottom of the graduated tube, a few drops of distilled water being first placed in the latter, and the mixture is rapidly agitated, to prevent the coagulation of the blood. The distilled water is then added drop by drop until the tint of the solution is the same as that of the standard, and the amount of the water added indicates the amount of hæmoglobin.

Fleischl's hæmometer consists of a stand to which is attached a reflector made of card-board. On the under surface of the plate there are two grooves, into which slides the frame, holding in position a wedge-shaped glass colored red, the intensity of the hue being graduated from zero to one hundred and twenty degrees. The frame is moved by means of a thumb-screw so that when it is operated the tinted glass passes beneath one of the compartments of the comparing vessel. The horizontal projection of the partition of this vessel should fall directly upon the outer edge of the glass wedge when the instrument is properly adjusted. In operating the instrument, care should be taken to have everything perfectly clean. Accompanying each apparatus are a glass pipette for dropping the water into the compartments, and several minute capillary tubes for securing the blood.

The compartments—that is, the blood and wedge compartments —are filled almost to the top with distilled water, and the vessel is placed *in situ*. The instrument should then be so arranged and the reflector so adjusted as to secure the full rays of light from either a candle, a lamp, or a gas-flame. Before securing the blood, the tip of the middle finger of the left hand should be carefully cleansed and dried. The automatic blood-pipette, with a capacity of six and a half cubic millimetres, and about eight millimetres long, to which is attached a frail wire for its manipulation, should always be greased, to prevent the blood from adhering to its sides. This is dipped into the blood sideways, to facilitate the flow into the tube: the greatest accuracy is essential to the correctness of the test. With as little delay as possible the tube is then placed into the blood compartment and its contents allowed to escape, aiding by gently moving the tube back and forth along its own axis. The diluted blood remaining in the tube is then washed out by means of the pipette and allowed to flow into the compartment. This is filled, as is the wedge compartment, with distilled water, care being taken not to allow the fluid in the two chambers to run together, and that the upper surface of the water is perfectly level, neither curved nor concave.

The blood is now ready for examination. In looking at the compartment the eyes should be shaded, that the direct rays of light may not cause error in the observation. The thumb-screw is turned, which slowly moves the wedge from right to left; this movement is continued until the eye can perceive no difference in color between the two compartments: should the difference be imperceptible for a considerable distance, then the point at which the color appears lighter and that at which it appears darker should both be noted and the mean ascertained. The number of degrees—that is, the percentage of hæmoglobin as compared with healthy blood, which is taken as one hundred—will be found on the movable slide.

Another and easier method of estimating the hæmoglobin is by taking the specific gravity of the blood. This is most readily done by Hammerschlag's method. It consists in mixing in an ordinary urinometer glass such quantities of chloroform and benzol as to mark 1059, the specific gravity of normal blood. A drop of blood in a pipette is blown into this chloroform-benzol mixture, and does not mix, but is seen to float. If it sink, add chloroform, a few drops at a time; if it rise to the top, add benzol until the drop of blood remains stationary in the body of the liquid, indicating that it has the same specific gravity as that of the whole mixed fluid. The specific gravity is then taken with the urinometer.

From the specific gravity of the blood we can deduce the percentage of hæmoglobin. Here is Hammerschlag's table:

Specific Gravity.	Hæmoglobin.	Specific Gravity.	Hæmoglobin.
1033–1035	= 25–30 per cent.	1048–1050	= 55–65 per cent.
1035–1038	= 30–35 "	1050–1053	= 65–70 "
1038–1040	= 35–40 "	1053–1055	= 70–75 "
1040–1045	= 40–45 "	1055–1057	= 75–85 "
1045–1048	= 45–55 "	1057–1060	= 85–95 "

In computing the hæmoglobin from the specific gravity of the blood, we must bear in mind that it varies considerably in dropsies, and that the weight of the leucocytes causes it always to be relatively higher in leukæmia.

A hæmoglobinometer based on the principle of the comparison of a thin film of undiluted blood, illuminated by candle-light, with a graduated color scale, has been invented by Arthur Dare,[1] and furnishes a rapid method of hæmoglobin estimation.

More important even than estimating the number of the corpuscles

[1] Philadelphia Medical Journal, April, 1900.

or the amount of hæmoglobin is the microscopical study of the blood, both in a fresh and dried state, and especially with the aid of stains. A drop of blood, taken from the tip of the finger or the lobe of the ear, is allowed to fall on a slide, and a cover-glass is placed over it. This answers for the study of the ordinary character of the red corpuscles, of the leucocytes, and of malarial parasites. But for finer study preserving fluids must be used, which are neutral diluting fluids, unstained or stained. Of the neutral unstained solutions those of Gowers, or Hayem, are mostly used. Gowers's solution consists of sodium sulphate, 104 grains; acetic acid, 1 drachm; distilled water, 4 ounces; Hayem's solution, of perchloride of mercury, 0.5 gramme; sulphate of sodium, 5 grammes; chloride of sodium, 1 gramme; distilled water, 200 grammes. Another diluting solution much employed for clinical purposes is that of Toisson, which, as it is colored, is especially valuable in enabling us to distinguish the leucocytes, which it colors blue, from the red blood-corpuscles, and to determine their relative proportion. It consists of glycerin (neutral), 30 cm.; sodium sulphate, 8 grammes; sodium chloride, 1 gramme; methyl-violet, 0.025 gramme; distilled water, 160 cm.

To obtain permanent preparations, and for purposes of greatest accuracy, the examination of the blood in films, especially in stained blood films, is necessary,—a method which we chiefly owe to Ehrlich. Blood films are usually prepared by allowing a drop of blood to fall on a perfectly clean cover-glass, to cover it with another, and then gently slide one over the other. The film dries in a few seconds, or rapid drying can be insured by swaying it in the air, or heating it over an alcohol lamp or for ten minutes in a dry heat sterilizer at a temperature from 100° to 150°. Immersion for about half an hour in equal parts of ether and absolute alcohol, as advised by Nikiforoff, is an excellent method for fixing a blood-film.

But staining may be essential, and this is done chiefly by aniline dyes. These are classified by Ehrlich as acid, basic, and neutral; and especially in studying leucocytes we make the greatest use of this division. The chief acid stain is eosin; methyl-green or methylene-blue represents the basic stains; neutral stains are a mixture of both; for instance, acid fuchsin with methylene-blue or green.

One of the most generally used stains is Ehrlich's triple stain; it consists of a saturated watery solution of orange G, 24–27 cc.; acid fuchsin, 16–33 cc.; methyl-green, 25 cc.; then add water, 60 cc.; absolute alcohol, 40 cc.; glycerin, 20 cc. The mixture should stand for one or two weeks before being used. Preparations ought to be exposed to the stain for several hours, having been previously thoroughly

heated: and to be preserved should be washed, dried, and mounted in Canada balsam. The modification of the Ehrlich stain, known as the Ehrlich-Biondi stain, is also much employed.

Stains are used as a means of classifying the leucocytes. Those containing granules that stain deeply with eosin or other acid aniline stains, and show as coarse, prominent granules, are called *eosinophiles*. Cells with fine granules which stain with basic aniline dyes, as with methylene-blue, are *basophiles*. Granules which stain with a mixture of basic and acid stain, as acid fuchsin and methylene-blue, are *neutrophiles*. They are also very conveniently stained by Ehrlich's, or Ehrlich-Biondi's, triple stain, and the granules are then violet or lilac, unlike the red or brownish-red coarse granules of the eosinophiles. By Ehrlich's stains the nuclei of the leucocytes are stained greenish blue.

In the minute study of the blood we pay close attention to its three elements, the red corpuscles, or erythrocytes: the white corpuscles, or leucocytes; and the blood-plaques, or blood-plates.

Red Corpuscles.—The red corpuscles are of various sizes. They have, according to Hayem, a mean diameter of 7.5 micromillimetres, the micromillimetre being $\frac{1}{1000}$th part of a millimetre: their color is due to hæmoglobin. Prolonged fatigue and menstruation diminish them. Their size varies much in disease. We may find many dwarf corpuscles or *mycrocytes*, having a diameter of from three to six micromillimetres, or numerous giant-cells, or *megalocytes*, with a diameter from nine to fourteen micromillimetres. In the latter, the amount of hæmoglobin is increased, and, in consequence, where they abound, as in severe anæmias, there is a high-color index.

The red corpuscles in disease not only undergo changes in size but in form. They lose their disk shape, and show irregular thickenings and projections at their borders, forming the so-called *poikilocytes*, common in, but not characteristic of, pernicious anæmia, and to be regarded essentially as a sign of degeneration. So, too, according to Ehrlich, is it a sign of degeneration or of death of the corpuscle, when with stains of eosin and hæmatoxylin the red corpuscles become violet or purple instead of pink or red. Where the corpuscles are found to be very pale or colorless, it is a proof of a low state. These "shadow corpuscles" are especially seen in protracted typhoid fever and where the blood is undergoing destruction and its hæmoglobin has been liberated from the red blood-cells.

A very striking change in the red corpuscles is their *nucleation*. This is never normal in the adult except in the immature red corpuscles in the bone-marrow, and is best seen in dry films stained with

DESCRIPTION OF PLATE V.

RED CORPUSCLES AND LEUCOCYTES.

The specimens were prepared by Dr. Boston, Bacteriologist to the Clinical Laboratory of the Pennsylvania Hospital, from cases chiefly of anæmia, pernicious anæmia, and leukæmia; they were drawn by Mr. Louis Schmidt from Queen Microscope, Obj. $\frac{1}{12}$″ oil immersion, eye piece 2, tube length 160 mm., and exhibit the effects of different stains.

The Red Blood-Corpuscles.—The preparations are stained with eosin and hæmatoxylin.

The first group represents *normal blood-cells* and shows a slight variation in their size; next comes a group of *microcytes*, of which two are deeply stained, the so-called Eichhorst corpuscles. Following, in the same line, are a number of large red corpuscles, or *megalocytes*, the two on the right showing some degree of vacuolation.

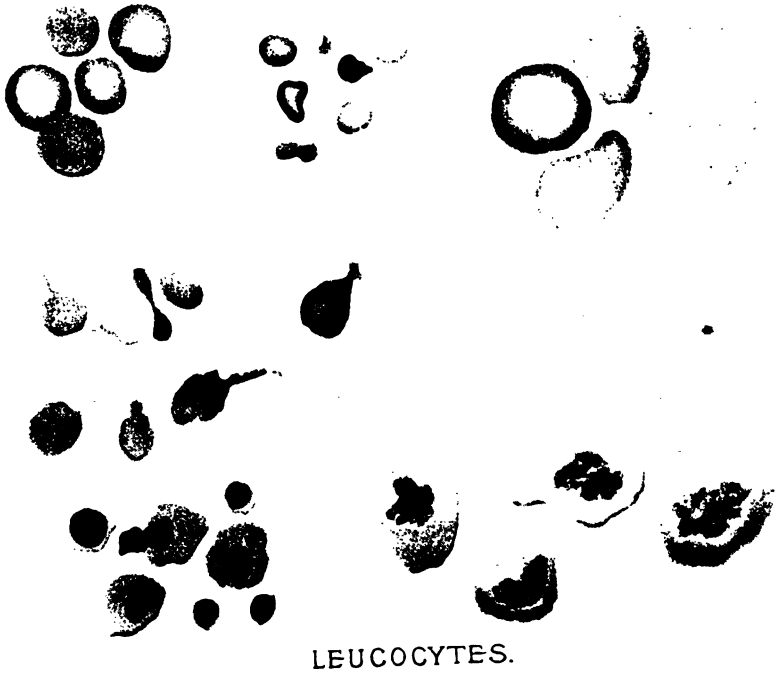
The second line begins with a group of *poikilocytes*, of various size, shape, and color; next is a group of pale or shadow corpuscles, followed by nucleated corpuscles of about the normal size,—*normoblasts*. The number of nuclei varies; in some the nucleus is partially extruded. The smallest elements shown are *microblasts*. Next will be found a number of *megaloblasts*, or large nucleated red corpuscles.

Leucocytes.—The first group shows three *small lymphocytes*, the second two *large lymphocytes*, all stained with Ehrlich's tri-stain. Following is a group of four polymorphonuclear *neutrophiles*. The first two are stained with Ehrlich's tri-stain; the other two with Ehrlich-Biondi stain, exhibiting fine neutrophilic granules.

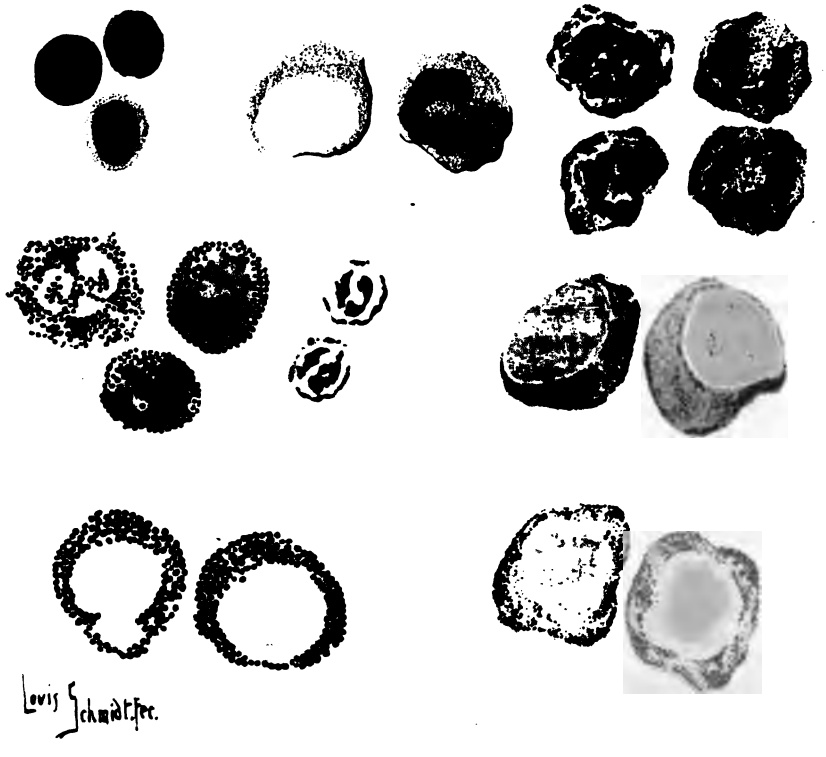
In the next line are shown normal *eosinophilic cells*, of two or more nuclei. The protoplasm contains large granules deeply stained with eosin. Next are two mast cells, stained with Ehrlich's stain. A group of *myelocytes*, stained with Ehrlich's stain, completes this line. Immediately below, on the last line, is shown a group of the same cells stained with Ehrlich-Biondi stain, as is also the group in the lower left-hand corner, showing marrow-cells containing eosinophile granules, or eosinophile myelocytes.

PLATE V.

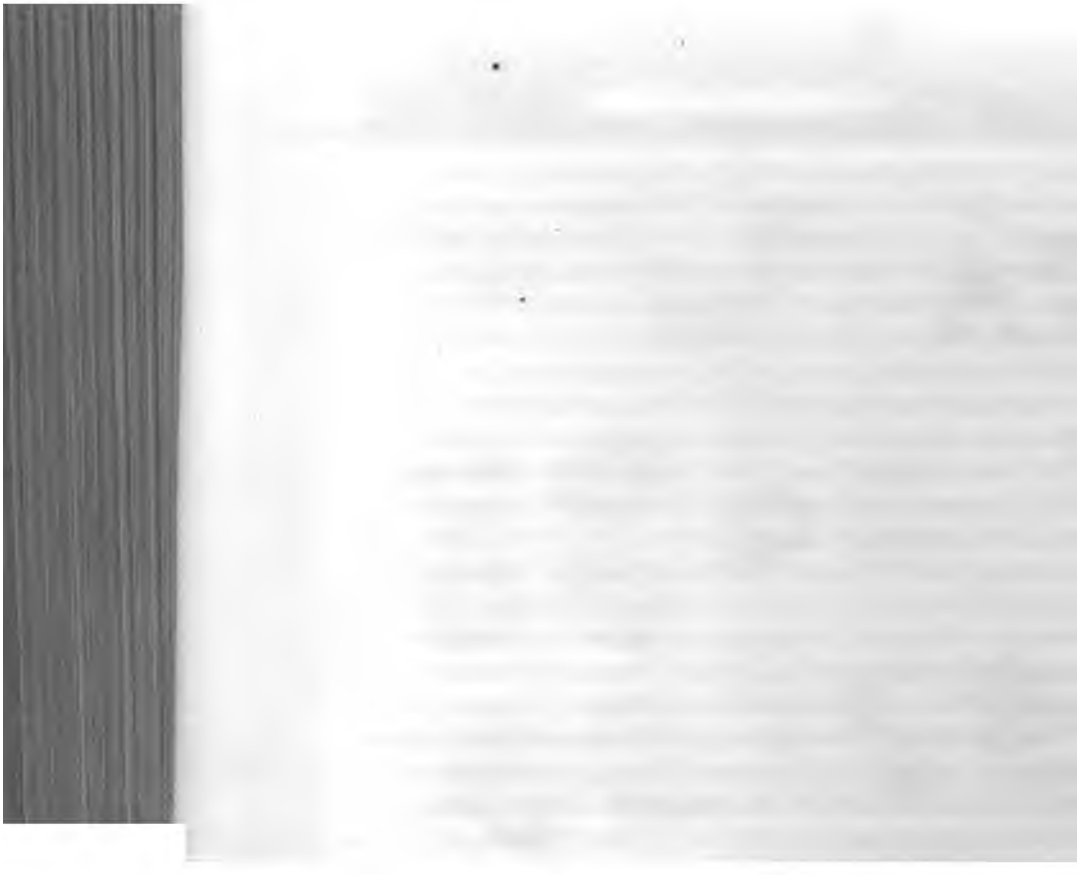
RED BLOOD-CORPUSCLES.



LEUCOCYTES.



Louis Schmidt, fec.



eosin-hæmatoxylin, or methylene-blue; the stain of the nucleus is especially deep. The nucleated blood-corpuscles are termed, according to their size, normoblasts, microblasts, and megaloblasts. The *normoblasts* are of the same size as the ordinary red corpuscle, and usually have a single nucleus which stains deeply; their outline is often irregular. They are a sign of new formation of blood, of an attempt at regeneration of the blood from the marrow, and may occur in crops, the "blood crisis" of Van Noorden. The normoblasts are found in marked anæmics.

The *microblasts* are nucleated red corpuscles, of smaller size than the normal corpuscles. Their occurrence is comparatively infrequent. The *megaloblasts* are much larger than the normoblasts, being from ten to twenty micromillimetres in diameter. The nucleus is very large, and takes a pale stain; the protoplasm around it stains deeply with eosin. Megaloblasts are never found in healthy blood; they indicate an abnormal state of the bone-marrow. They are a sign of degeneration, and are of grave import when in large numbers. Both normoblasts and megaloblasts may become poikiloblasts.

The red corpuscles stain especially with eosin; cells that stain with several colors from the same mixture, as with the Ehrlich-Biondi stain, becoming purple or gray or brownish in spots, are called *polychromatophiles*. They are especially met with in pernicious anæmia.

When blood has been standing for a short time the corpuscles form in *rouleaux*, and a fine *net-work of fibrin* is also seen. Rouleaux and the net-work of fibrin both show generally more markedly and quickly in inflammatory conditions. Decided net-works are also met with in many infectious diseases. Absence of rouleaux-formation never exists in health.

Leucocytes.—The white blood-corpuscles, or leucocytes, are pale, homogeneous or slightly granular, spherical cells, devoid of hæmoglobin. They are larger than the red corpuscles, but in number are few compared with these; ten thousand to the cubic millimetre is the normal limit. They increase after a meal and during pregnancy, and are numerous in the newly-born and in infancy. They contain one or several nuclei, are mostly amœboid, and some of them possess the power of attacking and digesting bacteria, therefore are "phagocytic." The leucocytes are variously affected by aniline dyes, as has been already explained, but are stained violet or lilac. Among the stained leucocytes the *eosinophile* cells are very important. These are actively amœboid. They are increased in asthma, in lithæmia, in affections of the liver, in trichiniasis, and often in spleno-medullary

leukæmia; they are diminished in influenza, in malignant tumors, in sepsis.

The chief forms of leucocytes in normal blood are the small uninucleated leucocytes, the large uninucleated leucocytes, and the multinucleated leucocytes. The first of these, also called the *small lymphocytes*, are estimated by Stengel at twenty-five per cent.; the large uninucleated or hyaline cells at three to six per cent.; the multinucleated neutrophile cells at sixty-five to seventy-five per cent.; the eosinophile cells not above three per cent. Cabot gives similar proportions, but adds "mast cells." These are his figures: small lymphocytes, twenty to thirty per cent.; large lymphocytes (same in structure, only larger), four to eight per cent.; polymorphonuclear neutrophiles, sixty-two to seventy per cent.; eosinophiles, one-half to four per cent.; "mast cells," one-fortieth to one-half per cent. Some observers describe separately, as a *transitional* or *intermediate* form, the large uninucleated leucocytes in which the nucleus is indented or horseshoe-shaped.

The small lymphocytes are about the same size as the red corpuscles; there is extremely little protoplasm, and they are not amœboid or phagocytic; the large multinucleated leucocytes are considerably larger; they are both actively amœboid, phagocytic, and neutrophilic, and the granules do not stain thoroughly except with triple stains like Ehrlich's. The so-called "mast cells" occur in health in only very small numbers. They are large, having a diameter of twenty micromillimetres or upward, and are coarsely granular. They stain with basic dyes, with dahlia or methylene-blue, are therefore basophilic, but do not show themselves with Ehrlich's triple stain. They get into the blood chiefly from the connective tissue. There has been some doubt as to whether they are not pathological; there is none as regards the *myelocytes*, or marrow-cells. They are very large cells with a pale nucleus, which with Ehrlich's stain is seen as a pale-stained nucleus nearly filling the cell; the protoplasm contains fine granules. The myelocytes are found in various intoxications, in myxœdema, in syphilis, but in large numbers only in medullary or spleno-medullary leukæmia.

Blood-Plates.—These, discovered by Hayem, and called by him hæmatoblasts, are small round or oval bodies of faintly yellow color, and very adherent. They may be seen in fresh blood, when immediately examined. They are smaller than the red corpuscles, colorless, and very cohesive. They are best studied with Hayem's solution, or a one per cent. solution of osmic acid; they stain faintly with aniline dyes, and number about two hundred thousand to the cubic

millimetre. They are observed to be increased in anæmias unaccompanied by fever, and after loss of blood; they are diminished in cachexias, particularly in cancer, protracted typhoid and typhus fevers, in erysipelas, and in all infectious fevers with high temperatures.

It is often a matter of great convenience to represent the blood-examinations graphically. An excellent chart for this purpose is in use at the Johns Hopkins Hospital. Fig. 72 shows it, and the manner in which the record is made.

FIG. 72.

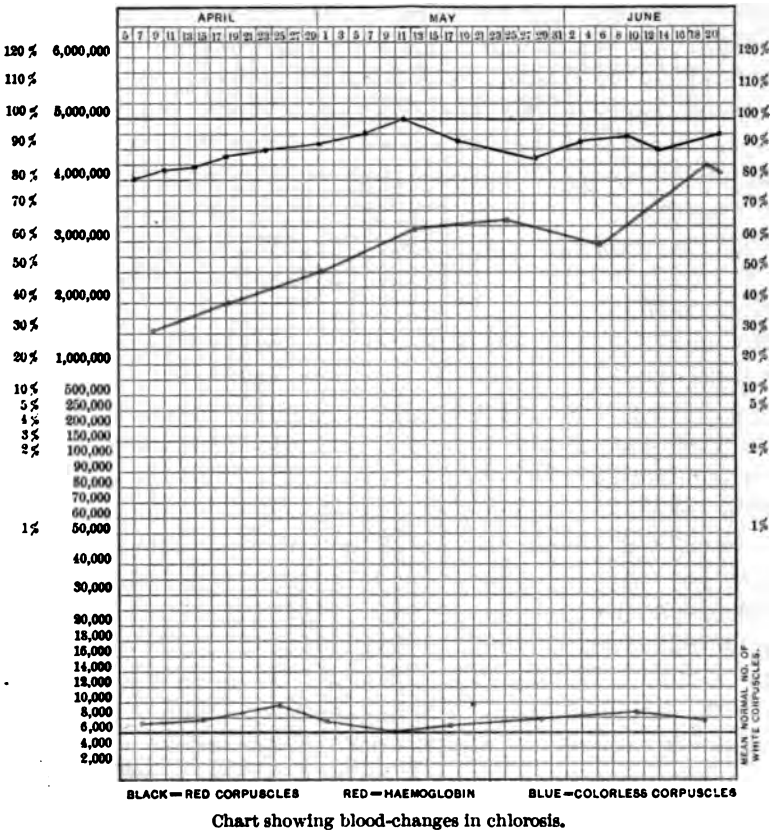


Chart showing blood-changes in chlorosis.

Anæmia.—Poverty of blood is met with as a consequence of profuse or frequently recurring hemorrhages, of insufficient nourishment, of affections which prevent the nutriment taken from being properly absorbed or assimilated, of disease of the blood-making organs, and of profuse chronic discharges, which drain the blood of many of its important elements, and especially of its albumin. Besides these causes of anæmia, we find it occasioned by particular

poisons, as by malaria; by syphilis; by uterine complaints; by the absorption into or the retention of noxious ingredients in the blood; by rapid destruction of the red corpuscles, as in fevers; and as consecutive to malignant growths, and organic diseases in general. Again, it is sometimes encountered without our being able to trace it to any obvious source. This is the so-called primary anæmia, as distinguished from the instances in which the anæmia is symptomatic of some disease, or secondary. But under all circumstances, except in the anæmia after hemorrhage, where all the constituents of the blood are diminished together, we have to deal with a blood deficient in red corpuscles, and the corpuscles are often badly shaped, and shrunken at their edges. In extreme anæmic conditions, large and giant corpuscles are common; the leucocytes and blood-plates are not altered, or are somewhat diminished. In secondary anæmia there is more apt to be an increase of leucocytes, the red corpuscles are dwarfed, and there are normoblasts rather than megaloblasts. The hæmoglobin may be increased relatively, or it may be diminished, or it may not be materially changed. Anæmia begins with four million corpuscles to the cubic millimetre.

Whatever may have given rise to the anæmia, the manifestations of the disorder when well marked are much the same. The patient is weak and pale; his lips and tongue have lost their red color; the eye is pearly; his pulse is feeble, and generally accelerated; the appetite is deficient or depraved; the bowels are apt to be costive. Yet persons, who are apparently well nourished and are not pale, may have deficiency of red blood-cells and of hæmoglobin. Exercise induces great fatigue, shortness of breath, and palpitation; and the disturbance of the heart may be associated with cardiac murmurs or with blowing sounds in the cervical veins, and is at times so persistent as to lead to structural changes. In some cases, we meet among the symptoms with obstinate headache and with dropsy, and in many with a persistent pain in the left side, in the region of the spleen.

Anæmia may be owing to the presence of parasites, such as intestinal worms. The very marked form which is common in Egypt is that due to *anchylostomiasis*. The *anchylostomum duodenale* is taken into the body in the muddy water, or by eating earth containing the embryos of this worm. Anchylostomiasis is an insidious, wasting disease, characterized by progressive anæmia and by digestive and nervous deterioration, occurring chiefly in earth and brick laborers of warm climates, caused by the presence in the duodedum and jejunum of a blood-sucking, rhabditic, nematode worm. The blood shows

great diminution of red blood-corpuscles, reduction of hæmoglobin,[1] megaloblasts, indeed all the signs characteristic of pernicious anæmia; but, as in bothriocephalus latus, these disappear with the expulsion of the parasite.

Chlorosis.—Here the pallid, wax-like countenance, the very pale lips, and the pearly eye afford unmistakable evidence of the deterioration of the blood, consisting chiefly in great deficiency of hæmoglobin, which is usually much more marked than the reduction in the red corpuscles; these, indeed, may be of almost normal amount. The corpuscles are pale. The smaller corpuscles, the microcytes, generally abound, and nucleated red cells are not infrequent; the leucocytes are not affected. Lloyd Jones[2] regards the disease as an exaggeration of a change which occurs in the blood of the healthy female at puberty, and which leads to an increase of the amount of blood-plasma associated with a diminution of the amount of hæmoglobin. Meinert has noted, in cases of chlorosis, gastroptosis combined with enteroptosis and occasionally movable kidney, and, as these displacements are produced by wearing corsets, his deduction is that chlorosis is due to faulty wearing-apparel. Chlorosis may also attend the absorption of ptomaines from the intestines in habitual constipation. Considerable stress has been laid on the fact that in chlorosis there is a greater tendency to inflammation of the optic nerve and retina than in pernicious anæmia, while the tendency to retinal hemorrhage is considerably less.[3]

The complaint is especially encountered in young women, and is, as a rule, associated with amenorrhœa. Indeed, many restrict the term to the obvious anæmia combined with suppression of the menses, so often affecting girls about the age of puberty. In pure chlorosis, organic diseases of the gastro-intestinal apparatus of the spleen and lymphatic glands, or of the lungs and kidneys, are absent; the temperature shows a slight rise; the nutrition of the body is fairly well kept up; the urine is pale and abundant, containing but a small amount of phosphates. Forchheimer finds a diminution of urobilin in the urine, which he regards as of considerable diagnostic importance. The nervous system is irritable. Pigmentation about the second joints of the fingers, on their dorsal surface, has been noticed.[4] Sometimes these symptoms of chlorosis happen before puberty; or

[1] Sandwith, Proceedings XI. International Med. Congress, Rome, 1894.

[2] Brit. Med. Journ., July, 1894.

[3] Stephen Mackenzie, Sajous's Annual, 1895, vol. i. L. 10.

[4] Bouchard; also Pouzet.

there are relapses of the malady in middle age. Boys about the age of puberty may also develop the manifestations of chlorosis. Virchow has pointed out the frequent association of chlorosis with narrowing of the aorta and of the great arteries, and such cases are distinguished by obstinate relapses. There is a variety of chlorosis in connection with tubercle, at times preceding it. Chlorosis as well as anæmia may be associated with nasal hypertrophies or *adenoid vegetations* in the vault of the pharynx,[1] and be relieved by their removal. Both the corpuscles and the hæmoglobin may be decidedly decreased in consequence of surgical shock.[2]

Fever may occur in chlorosis, though to but slight degree. Jaccoud attributes it to *anoxæmia*, the deficiency of oxygen in the blood acting as a stimulant to the calorific centres. Fever may be also due to local causes, such as phlegmasia alba dolens.

Pernicious Anæmia.—This is an extreme anæmia advancing steadily, or with remissions, towards a fatal ending; yet no certain cause can be detected for the profound and disastrous alteration the blood is undergoing. To pernicious anæmia belong most of the cases of "essential" or "idiopathic anæmia."

The disorder is most frequent in women, and has been especially observed in childbearing women after several pregnancies; still, it also often happens in men, especially before the age of forty. It sometimes seems to have its origin in long-continued dyspepsia or diarrhœa, and atrophy of the gastric tubules; or to arise after protracted hemorrhages or incessant worry,—after, indeed, slowly but steadily acting debilitating influences; and it has been noted to arise after nervous shock, or to be of parasitic origin, and due to worms. sometimes to a tape-worm,—bothriocephalus latus.[3] But in the majority of instances it originates seemingly without cause, and, although it has periods of deceptive improvement that may last for months. or, as I have known, even for a year, it progresses relentlessly towards a fatal issue.[4] It is true that some cases of recovery have been recorded; but of these it is not quite certain that they presented all the characteristic symptoms.

There is an insidious beginning, except at times when the anæmia

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[1] F. Oppenheimer, Berl. klin. Wochenschrift, Oct. 3, 1892; Sajous's Annual of the Universal Med. Sciences, vol. iv., 1894.

[2] Joseph Leidy, Jr., Transactions of the College of Physicians, Phila., 1893, vol. xv. p. 242.

[3] Schmidt's Jahrb., i., 1891; also *ibid.*, No. 10, 1887; and Berl. klin. Wochensch., No. 40, 1886; also Deutsches Arch. für klin. Med., Bd. xxxix.

[4] See also case with remissions in Schmidt's Jahrb., No. 4, 1882.

develops itself in the pregnant state. Pale tongue, bloodless lips, pearly eye, becoming paler, more bloodless, more pearly, from week to week; breathlessness; palpitation of the heart, especially on exertion; weak digestion; constipation, or constipation alternating with diarrhœa; loud systolic murmurs in the heart, and venous hum in the jugulars; vertigo, a marked lemon-colored hue of the skin about the large joints, at times jaundice; finally extreme exhaustion, sluggishness of mind, fainting-fits, and dropsy, without persistent albumin in the urine, or disease of the liver, or enlargement or valvular disease of the heart, to account for it,—are the prominent symptoms. In the later stages, too, hemorrhages from the nose and from the gums are not uncommon; and hemorrhages from the uterus or from the kidneys, or into the skin and into the retina, may be also noticed; the latter especially are very frequent. Yet, notwithstanding all these grave signs, the body appears well nourished; there is certainly no decided emaciation, except in instances in which fever is more than commonly marked. Now, fever is a significant feature of progressive pernicious anæmia; it has been present in every case that I have met with. It is not an early symptom, belonging to the full development or to the latter part of the disease. It is of very irregular type, and not of high intensity, the temperature rarely exceeding 103° F. It is apt to be continued, or to show occasional exacerbations, followed by remissions, the febrile state lasting for days, or even for a week or two at a time; then there are periods of shorter or longer duration when it wholly disappears, to come on again in an outbreak attended with all the usual signs of a febrile paroxysm for which no cause is apparent. Towards the end of the case it is not unusual for the anæmic fever to have entirely ceased, and for the temperature to have fallen below the normal standard. Pernicious anæmia may run an acute course.

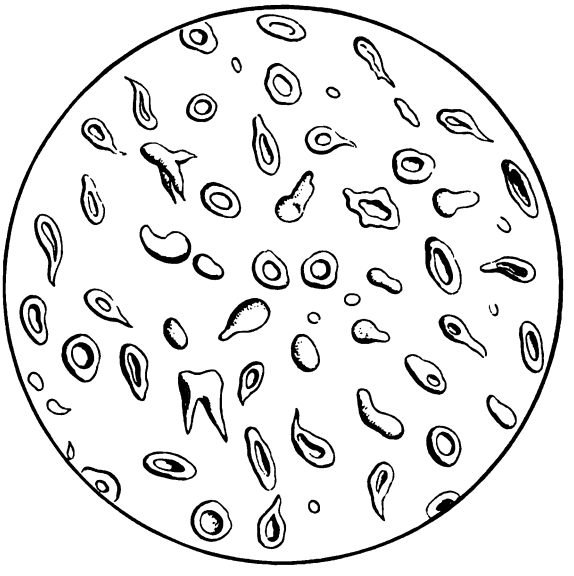
In this perilous malady the red globules are strikingly diminished in number,—to about a million and a half or less; the white corpuscles are not relatively altered, or they may remain normal, and seem to be increased, because the red globules are much fewer. Towards the end there may be, indeed, a true leucocytosis.[1] The hæmoglobin, while, in the whole amount, markedly lessened, is in the individual corpuscles generally increased,[2] the leucocytes are normal or diminished, the pale hæmatoblasts are diminished and may quickly assume irregular shapes. The red corpuscles are generally increased in size. Eichhorst regards as a characteristic change that the blood

[1] Stengel, Twentieth Century of Medicine.

[2] Hayem, Du Sang, Paris, 1889.

contains also a quantity of ill-developed, small, spherical, highly colored red corpuscles. But these are not pathognomonic; for they have been found by Cohnheim in medullary leukæmia, and by Greenfield in lymphadenoma; on the other hand, they are frequently absent. Besides this there are giant-cells of irregular shape, on which Hayem[1] lays great stress, also many very large normal-looking red corpuscles, some of which are, however, nucleated megaloblasts. Nucleated red corpuscles were detected in the blood of all the patients examined by Howard:[2] the blood seems to revert to a lower type.

FIG. 73.



Blood in pernicious anæmia, illustrating the irregularly shaped blood-cells (Poikilocytosis).

This has been also insisted upon by Henry. Stengel regards the nucleated red blood-corpuscles as a constant feature. The accompanying cut (Fig. 73), from a well-marked instance of the disease, shows the irregular shape of the corpuscles and their varied size and appearance; some are nucleated.

Of the real cause of the disease we are in ignorance. No constant lesion of the blood-making glands has been found; but everything points to excessive blood-destruction. The structure of the spleen and of the lymphatic glands is not altered; the marrow of the bones

[1] Op. cit.

[2] Montreal General Hospital Reports, vol. i., 1880.

may or may not be.[1] Hunter[2] has brought forward strong proof that the characteristic anatomical change is the presence of an excess of iron in the liver, the seat of disintegration of the corpuscles being chiefly in the portal circulation. The existence of some toxic substance in the circulation is highly probable, and by some this is thought to come from intestinal autointoxication. Diminished or faulty hæmogenesis may also exist and contribute to the anæmia, as insisted upon by Van Noorden.[3]

There is also a close association with structural disease of the brain and cord. Degeneration of the lateral pyramidal and lateral cerebellar tracts, and especially of the posterior columns of the spinal cord, has been found. But the question of the relation of these degenerative changes to pernicious anæmia is unsettled. They have been studied by Lichtheim,[4] by Van Noorden,[5] by Bowman,[6] and more recently by Burr,[7] who found the gray matter seldom even slightly involved. He favors the view that both the anæmia and the cordal lesions are due to a common cause,—a poison or poisons, as in diphtheria or ergotism, and Lichtheim's opinion is similar.

The diagnosis of pernicious anæmia is never an easy one, for it is difficult to be quite certain that no latent organic disease exists which would account for the progressive alteration of the blood. Indeed, without the microscopical features of the blood a diagnosis is impossible, and ought not to be attempted. While no one element is characteristic of pernicious anæmia, this state of the blood warrants it: red cells extremely low, two million or lower; some, but not marked, increase of the white cells; hæmoglobin variable, relatively increased; high color-index; many large and many very irregularly shaped red corpuscles (megalocytes and poikilocytes); red corpuscles, whether of irregular shape and size or not, frequently nucleated (poikiloblasts and megaloblasts).

With reference to diseases likely to be confounded with pernicious anæmia, I have more than once known obscure *organic disease of the stomach*, especially gastric cancer, where the tumor could not be discerned, or *contracted kidney*, with but little albumin in the urine, and where the anæmia was marked, to be regarded as a typical illustration

[1] Pepper, Amer. Journ. Med. Sci., Oct. 1875; see also Cohnheim, Virchow's Archiv, Bd. lxviii., and Waldstein, Arch. f. Path. Anat., Berlin, 1883, xci.

[2] Lancet, London Practitioner, Aug. 1888.

[3] Quoted in Sajous's Annual for 1895, vol. i. L. 8.

[4] Congress für Innere Medizin.

[5] Charité Annalen, 1891.

[6] Brain, 1894.

[7] University Medical Magazine, April, 1895.

of the malady, until the autopsy revealed the true cause of the fatal exhaustion. With reference to the former affection the error is all the more likely to happen because symptoms of gastric disorder are usual in progressive anæmia; with reference to disease of the kidney the misleading part is that a trace of albumin is occasionally present in progressive anæmia. But it is not persistent, is associated with marked evidence of urobilin and with increase of uric acid, and a microscopical examination of the urine will tell us the real extent of kidney affection. The cachectic pallor of subjects of *malignant disease* may be mistaken for the lemon- or straw-colored appearance of the skin in pernicious anæmia, and the anæmia is often pronounced, and poikilocytes and normoblasts are common. But megaloblasts are few, and this, Cabot states, is a valuable distinguishing mark from pernicious anæmia.

Diseases of the heart may be held to be pernicious anæmia. A fatty heart, in an elderly person, with or without valvular disease, with failure of strength, and with the peculiar pallid, sickly look occasioned by the malady, may mislead. But the long duration of such cases, and the absence of fever, are strong points in the case. Indeed, the error is apt to be the other way,—that, overlooking the symptoms of profound anæmia and general failure, we regard the murmurs and the other cardiac symptoms which are associated with the fatty heart of pernicious anæmia, a very commonly coexisting lesion, as pointing to a disease of the heart alone. The physical signs will not always assist: the murmurs may be very distinct and loud.

A number of trophic and vascular disturbances have followed surgical extirpation of the thyroid gland, and have been also noticed after atrophy of the gland has occurred. To this condition the name of *cachexia strumipriva* has been given. It is distinguished from anæmia by the occurrence of signs of myxœdema, often with cretinism and circulatory disturbances, with local asphyxia and transient or intermittent albuminuria. In some cases epilepsy is developed, in others pulmonary phthisis.

From the other diseases of the blood pernicious anæmia is distinguished by the special features of the blood already mentioned, particularly by the large and irregular forms of the blood-cells. Besides, it differs from *ordinary anæmia* by its relentless progress and the little influence the most nourishing diet and courses of iron have on it. Moreover, the distinctness of the cardiac murmurs, the slight emaciation, and the irregular outbreaks of fever are significant. The marked accessions of fever, the presence of dropsy, though moderate, the retinal extravasations, the other hemorrhagic symptoms, and the unyielding blood-change, separate pernicious anæmia from *chlo-*

rosis. The pernicious malady sometimes seems to develop out of a long-standing chlorosis, and then the grave symptoms just spoken of prove its supervention. The same grave symptoms happen also, at least the hemorrhages are as frequent, and the fever and dropsy may happen, in *leukæmia* and in *pseudo-leukæmia*. But the great increase in the white corpuscles, the tumefaction of the spleen, or the affections of other blood-making parts, distinguish the former malady; and pseudo-leukæmia, while the blood microscopically may be that of a severe anæmia, exhibits the enlarged lymphatic glands, their progressive invasion, the lymphoid tumors, the abdominal pains, and the steadily increasing emaciation so characteristic of the disease. Moreover, here the red corpuscles, as in ordinary anæmia, are usually smaller and paler than normal, and nucleated blood-corpuscles are rare, and not of giant size; and while the corpuscles are not markedly diminished in number, the hæmoglobin is strikingly lessened.

Leucocytosis.—Leucocytosis is an increased number of white cells over the normal amount as found per cubic centimetre. All the different varieties of leucocytes are increased, though in pathological states the increase shows chiefly in the polymorphonuclear cells. The leucocytes are increased after meals, and decreased by starvation. Cabot advises to examine the blood shortly before a meal, and preferably before breakfast. The leucocytes are in large numbers in the new-born, in pregnancy, and after parturition. Pathologically, we observe a marked increase of leucocytes after hemorrhages, especially in the polymorphonuclear cells; in a number of infectious diseases, such as in typhus fever, relapsing fever, erysipelas, diphtheria, pneumonia, scarlet fever, smallpox, cerebro-spinal meningitis, malignant endocarditis, in trichiniasis, in pyæmic and septicæmic states; and in all inflammatory conditions, including those of the skin. We also find leucocytosis in malignant disease; in gout and in lithæmia; in uræmia and other toxæmias. Some of the animal extracts, such as of the spleen and bone-marrow, produce it, and among drugs pilocarpine and antipyrine do so decidedly. On the other hand, in typhoid fever, in influenza, in measles, in German measles, in malaria, and in tuberculosis, there is no leucocytosis. It is difficult to determine at what actual number of white cells leucocytosis begins. Both Hayem and Stengel regard it as present when the leucocytes exceed 10,000 per cubic centimetre. Leucocytosis ends, according to some authors, at 70,000, to others, at 100,000, and we are then dealing with white blood, or leukæmia. Yet it is not so much the mere number of leucocytes as their alterations and their changed proportions that determine this.

Now it is always most important to study these alterations, and the proportion the various forms of leucocytes bear to each other. As already stated, the multinucleated cells are the ones that in leucocytosis, especially that of pathological states, are mainly increased; but in rickets, in syphilis, in cervical adenitis, in tumors of the spleen, in far-advanced cachexias, and after taking a course of thyroid extract,[1] the greatest relative increase may show itself in the lymphocytes. Cabot tells us that in obscure syphilitic cases the diagnosis may be made by the coincidence of lymphocytosis with increase of the eosinophiles. Myelocytes occur only in pathological conditions, such as in leukæmia and in grave anæmias, and markedly in those of syphilis and cancer.

In the study of alterations of the cells, we pay particular attention to their shape, granulation, and the nuclei. In addition to typical cells, we find in very marked instances of leucocytosis cells that are not typical, but appear like transition cells. In malarial fevers and cachexias, and in melanosis, the leucocytes are often pigmented.

A *decrease* in the number of leucocytes is found from starvation, in low fevers of long duration, and in pernicious anæmia. Diminution of the leucocytes is named *leucopenia*. The diminished number of leucocytes may not be real, but be owing to incarceration in the finer capillaries. This state has been called by Maurel *false hypoleukæmia*. The arrest of the white cells may be due either to vasomotor constriction of the small capillaries or to direct action of poisonous agents upon the leucocytes, giving them a spherical shape and sluggish amœboid movement.

Leukæmia.—This morbid state consists in a decided increase of the white corpuscles and a decrease of the red. Under the microscope the white globules of the blood, instead of bearing the normal proportion of about 1 to 500 of the red, are found in the proportion of 1 to 6, or even 1 to 0.5, and cases have been met with in which near the point of death the white corpuscles have been five times as many as the red. Besides the increase of white corpuscles and the diminution of the red, peculiar, colorless, shining, elongated octahedral crystals have been pointed out by Neumann and by Charcot. Haig has stated that the proportion of uric acid in the blood is increased in splenic leukæmia. Jaksch has shown that the blood is rich in peptone, although this substance is rarely met with in the urine in leukæmia. Mathes found deutero-albumoses in the blood and serum.

The abnormal condition exists in connection with hypertrophy of

[1] Perry, New York Medical Record, Aug. 1896.

the spleen, "splenic leukæmia," or of the liver; with other diseases of these viscera; and with various malignant or non-malignant affections of the lymphatic glands, "lymphatic leukæmia;" or of the thyroid body, especially with an increase of the cellular elements. But none of the blood-glands is so constantly and so markedly affected as the spleen. We have, too, a "myelogenous" or medullary form of leukæmia. In splenic leukæmia there are also very often marked marrow-changes; hence this is mostly described as splenic myelogenous leukæmia, or spleno-medullary leukæmia.

The disorder may occur at all ages; it is more common in men than in women. Leukæmia is consequent upon obstinate intermittents with decided enlargement of the spleen, syphilis, over-exertion, long-continued mental depression, chronic intestinal catarrh, and blows on the splenic region. The form affecting the marrow of the bones frequently results from injury to the bones. Ebstein reported cases of leukæmia following traumatism, but the causative relationship is not clearly made out. Yet in many cases of leukæmia no adequate cause can be detected. Its beginning is usually gradual and ill defined; sometimes it clearly follows other diseases. When fully developed, it occasions, besides the obvious pallor and the cachectic appearance, feeble heart action, exhaustion, diarrhœa, hurried breathing, hemorrhages from various parts, especially from the nose, profuse sweating, slight rise of temperature in the evening, increase of uric acid in the urine, fleeting abdominal pains, and dropsy dependent upon the enlargement of the spleen or of the liver, or upon the leukæmic new formations in the latter. In some cases a swelling of the glands on both sides of the throat, attended with inflammation of the mucous membrane of the mouth and pharynx, and followed by swelling of the axillary and the inguinal glands, precedes the enlargement of the liver and of the spleen.[1] Indeed, glandular tumors are often present; the glands of the groin are, as a rule, enlarged. There is disturbance of vision, connected with retinal changes, and in some instances deafness, and peritoneal or pleural inflammation, also melancholy. Pain in the bones, too, particularly in the sternum, is observed. The medullary or myelogenous variety is pre-eminently marked by pain, which is increased or developed by pressure over the sternum and ribs and over other affected bones.[2]

[1] Mosler, in Virchow's Archiv, xliii.; Dunn, Amer. Journ. Med. Sci., March, 1894, describes a case with growths in the orbits.

[2] Mosler, Berlin. klin. Wochenschrift, xiii., 1876; and Schmidt's Jahrb., No. 10, 1877.

The diagnosis of leukæmia is possible only by the microscopical examination of the blood, which detects the decided increase of the white corpuscles, and especially by studying the kinds of cells present in stained films of the blood. In the most common variety, *splenic* or *spleno-medullary* leukæmia, for in this combination it generally exists, we may be also able, even early, to discern the enlargement of the spleen, and to find the evidences of cachexia in the appearance of the patient, and in recurring epistaxis. But it is the microscopical examination of the blood alone which enables us to distinguish leukæmic swelling of the spleen from malarial or other affections. And to have a definite diagnostic meaning the white corpuscles must be decidedly and permanently increased and altered in shape. It is, indeed, not easy to draw a line between leucocytosis, however caused, and leukæmia. Anything above 100,000 white cells to the cubic centimetre is looked upon as exceeding the limit of leucocytosis. But it is the character of the elements of the blood, not the mere number of the white cells, that positively determines the diagnosis. The characteristic feature is the great preponderance of marrow-cells. These myelocytes are generally large, highly granular, often irregular, and, stained with aniline, they show themselves as the so-called eosinophile marrow-cells. Besides this, the eosinophonic cells are increased, as well as the lymphocytes; though this increase is very slight in proportion to the enormous number of the myelocytes. There are all forms of intermediate, irregular leucocytes. There is only a slight decrease in the number of the red cells, notwithstanding the enormous increase of the white cells, and among the red cells are many nucleated ones. As contrasted with leucocytosis, Hayem and Cabot lay stress on the fact that the large white cells are mostly not amœboid. These characters of the blood distinguish splenic leukæmia from splenic anæmia, from pseudo-leukæmia, and from malarial enlargement of the spleen. In all these, moreover, the leucocytes, even if increased, are not markedly so, are not abnormal in appearance, and the differential enumeration gives a wholly dissimilar result. The red corpuscles are much more apt to be decreased; there is, indeed, more or less anæmia. In *lymphatic leukæmia* there is marked swelling of the lymphatic glands, while the spleen is but slightly, or not at all, enlarged. The leucocytes are also markedly increased, but not to the extent found in splenic leukæmia, and they are almost entirely lymphocytes. Polynucleated leucocytes, so common in splenic leukæmia, are few, only about three per cent.; myelocytes are mostly absent; and so are nucleated red corpuscles. In the *medullary* form of leukæmia, rare except in combination with the splenic, there is

obvious abnormal condition of the spleen and the lymphatic glands, the blood shows marrow-cells in enormous numbers and in all stages of development, many very granular or undergoing multiplication by indirect nuclear division.

In comparatively rare instances leukæmia runs an *acute* course, varying in duration from one to nine weeks to its termination. It may be of the splenic, splenic medullary, or lymphatic variety. The disease generally sets in with chills, the fever is irregular, the spleen or the lymphatic glands enlarge, and a hemorrhagic tendency manifests itself. The blood condition is the same as in the chronic form, especially the lymphatic variety, and the small uninucleated leucocytes are immensely increased. It is almost invariably fatal. Bramwell[1] has reported a case recovering rapidly under quinine.

Lymphadenoma.—As regards the symptoms, the closest similarity to leukæmia is presented by the affection described as lymphadenoma, pseudo-leukæmia, or *Hodgkin's disease.* It consists in an enlargement of the lymphatic glands of the body, often with lymphoid growths in other parts, which soon becomes complicated with weakness and signs of cachexia, with diarrhœa, with dropsy, with cardiac palpitation, shortness of breath and attacks of suffocation, with tendency to profuse bleedings and to bedsores, and leads usually, in the course of not many months, or, at farthest, of a few years, to death. There is often a sense of fulness in the abdomen, attended with violent pains; the temperature in advanced cases shows mostly an evening rise. Some of the superficial lymphatics are first affected, others follow; the disorder then extends more decidedly, the spleen and the liver increase in size, other organs, too, may become involved, and lymphoid tumors develop in various parts of the body; but among the internal organs the spleen is the one most constantly disturbed.

The disease generally begins in the cervical glands; far less frequently does it show itself first in the inguinal or in the axillary glands; still less frequently in the bronchial or in other internal glands. The affection occurs much oftener in men than in women. It mostly happens in males between the ages of ten and thirty-five and of fifty and sixty; it is not very uncommon in young children. Its cause is unknown; it certainly has no definite connection with either scrofula or syphilis. In infancy the disease, as von Jaksch shows, occurs as a combination of a grave anæmia with marked leucocytosis.

The blood shows some deficiency in red globules, but otherwise no constant alteration. Slight increase of leucocytes has been occa-

[1] Anæmia, Edinburgh, 1899, p. 164.

sionally noticed, especially during the later stages; the white corpuscles are generally small and uninucleated or multinucleated. Myelocytes are absent. The hæmoglobin is reduced.

It is this difference in the state of the blood that makes the chief distinction between pseudo-leukæmia and *leukæmia*, in which there may be glandular enlargements. Rare cases of diffused *lymphatic cancer* closely resemble Hodgkin's disease; so closely that they are undistinguishable, except by the history of the case and by a microscopical examination of any of the tumors that may have been removed; the spleen is not involved, while the organs contiguous to the glandular cancer are likely to be more rapidly implicated. In *sarcoma of the lymphatic glands* the disease is at first strictly local, and then, if it spread, invades not the lymphatic tissues specially, but any part of the body; the enlarged glands do not move freely on each other as they do in lymphadenoma; and the blood-changes are those of a secondary anæmia. Perhaps the fact Cabot regards as of much value —that in cancerous anæmia the megaloblasts are always fewer in number than the normoblasts—may here prove of decided use. *Local gland lymphomas* are separated from Hodgkin's disease by their local character, by their want of extension, and by the absence of marked cachexia. *Scrofulous* or *tuberculous glands*, unlike lymphadenoma, enlarge rapidly, have thickened tissue around them, and are apt to undergo cheesy degeneration, or to soften and suppurate. Moreover, they are associated with the presence of tubercle bacilli, and mostly affect the submaxillary glands. The anterior cervical glands are the ones chiefly and primarily affected in Hodgkin's disease. In *splenic anæmia*, or *splenic pseudo-leukæmia*, as it is less appropriately called, we have the same condition of the blood as in Hodgkin's disease, save that there is mostly a much greater decrease in the red blood-corpuscles, and nothing absolutely distinguishes it except the absence of enlarged external lymphatic glands, and the more decided increase of the spleen, which, though greatly enlarged, is unaltered in shape. There may be some enlargement of the retro-peritoneal glands, and there is variable fever, as in other grave anæmias. In some cases of Hodgkin's disease fever is a prominent symptom, and this may be of intermittent type, giving rise to the belief that we are dealing with a *malarial affection;* recurring chills make error still more likely.

In the early stages of lymphadenoma a diagnosis is impossible, and we are at a loss to account for the increasing signs of cachexia, until the involvement of the lymphatic glands in rapid succession, and their quick growth, or the speedy formation of other lymphoid tumors

under the skin or in other parts of the body, clear up all doubt. There will also be great uncertainty in all those instances in which the growths happen first in internal glands or structures,—as in the bronchial glands and the mediastinum, producing severe bronchitis, extreme dyspnœa, and signs of venous stagnation in the veins of the upper part of the body; or as in the glands around the biliary ducts, giving rise to jaundice; or as in growths of the spinal cord leading to paraplegia,—until the external swellings explain the case. The kidney is not an organ that often suffers primarily; the occurrence of more than a mere trace of albumin shows that it has become implicated from parenchymatous changes or disseminate lymphoid growths. Lymphadenoma may run an *acute* course, with fever and marked hemorrhagic tendency.

Addison's Disease.—While seeking for the explanation of puzzling cases of anæmia, Addison discovered that a peculiar anæmia always occurs in connection with a diseased condition of the suprarenal capsules, and is characterized by distressing languor and great general prostration, remarkable feebleness of the heart's action, loss of appetite, obstinate vomiting, and a singular alteration of the skin. This consists in a dingy or smoky hue of the surface; or the color may be of a deep amber or chestnut brown, or the altered skin may have a bronzed tinge. The change of color begins on exposed parts, such as the face and neck and the back of the hands, and deepens first there; but we also soon find it marked in parts which are naturally the seat of much pigment, such as the axillæ, the groins, and the areolæ of the nipples. It is also marked around the umbilicus, on the penis, and on the scrotum, and is dependent upon a layer of pigment in the rete mucosum. There are also deposits of pigment on the lips and gums and other mucous membranes. The skin remains soft and smooth, and becomes in large portions uniformly discolored, gradually deepening, and often presenting a hue on the face and hands like that of a mulatto. Any irritation of the skin is followed by dark streaks. Discoloration in patches is both less constant and less significant than extensive alteration of hue; yet the darkening in undoubted cases may occur in patches, which are usually most obvious on the face or the superior extremities. The patient may seem at first sight to be jaundiced; but the pearly whiteness of the conjunctiva soon dispels such an idea. The nails are pale and bluish; the tongue may have patches of dark color; the body and breath at times exhale an offensive odor. The blood does not undergo any characteristic alteration. It shows a more or less marked decrease of the red corpuscles, without any change in the white. These are

sometimes found to contain black pigment granules. The hæmoglobin is but little, if at all, below the normal average.

With reference to the other symptoms, the most conclusive of them are remarkable prostration, generally without any marked waste of the body, feebleness of heart action and of pulse, and obvious anæmia. In most cases, but far from in all, these symptoms precede the discoloration of the skin; and they are not infrequently associated with pain in the back and with nausea and vomiting and attacks of diarrhœa, with breathlessness upon exertion, with vertigo, and with dimness of sight or impaired hearing. A peculiar odor of the body, like that perceived in the colored race, was observed in two cases placed on record by Mr. Hutchinson. In the later stages of the malady the temperature falls below the norm. The pulse, in place of being feeble, may be of strikingly high tension, owing to the absence of the secretion of the suprarenals, and we may recognize disease of these organs from this high tension even when no pigmentation exists, provided we are able to exclude other causes for a high-tension pulse.

Death may take place gradually from the constantly growing asthenia; or it may occur suddenly, and where the amount of prostration does not appear so excessive as to foreshadow it. According to the elaborate researches of Wilks, the destruction of the capsules is dependent upon a peculiar scrofulous degeneration; and this view of the tubercular nature of Addison's disease is now very generally held.[1] Should this prove to be correct,—should it appear, in other words, that the nature of the disease of the adrenals influences the symptoms more than the mere fact of their being diseased,—it would explain why in some cases of absence of the glands, or of their cancerous degeneration or suppuration, no signs of Addison's disease existed. Yet tuberculous disease of the adrenals, with tubercle bacilli in the caseous glands, has been found without bronzing of the skin.[2] Many of the symptoms of the fully developed malady may be due to the implication of the nervous branches, derived from the sympathetic and the pneumogastric, which go to the glands. Indeed, the idea of the primary seat of the disease in the abdominal sympathetic nerve is strongly advocated by some observers. Bramwell calls attention to the frequency of coexisting atrophy of the heart.

Now, in the diagnosis of Addison's disease the alteration of the color of the skin plays so important a part that we must inquire

[1] See, for analysis of cases, Gilman Thompson, Transact. of Assoc. of Amer. Physicians, 1893.

[2] As in the case of Ballenghien, Journ. des Sci. Méd. de Lille, 1888.

whether it or something very like it may not happen in other conditions. In persons *long exposed to the sun* a bronzing of the face and neck and arms occurs; but it is extremely uniform; there is a striking contrast between it and the parts that are not exposed, including such as we find greatly affected in Addison's disease, the flexures of the joint, the scrotum, the textures around the nipple and the umbilicus. Moreover, there is often robust rather than impaired health. In persons who, in addition to exposure, are of uncleanly habits and infested with vermin, especially in elderly persons, a discoloration of the skin happens at various portions of the body, often deepest on the chest, the abdomen, and the back, which is readily mistaken for the bronzing of Addison's disease. But in this *vagrants' disease* the discoloration is in the superficial, not in the deeper layers of the epidermis, and the dark cuticle is harsh and raised, not soft and smooth. Then alkaline baths and washing with soap will greatly diminish the deepened hue. A similar bronzing of long standing, though of doubtful origin, is sometimes met with.[1]

During *exhausting lactation*, or in *pregnancies* attended with much constitutional disturbance, there may be marked discoloration of the skin; yet it is not most obvious on the face, and the circumstances of the case are important aids in the diagnosis. So is the history in those instances in which a bronze hue is *hereditary*,[2] or in which a very deceptive discoloration attends *tubercular peritonitis* or *chronic diseases of the liver*, especially cirrhosis; or follows *yellow fever*, or the *malarial fevers*. In these diseases, too, the discoloration is not so great, and it is not marked at the sites most affected in Addison's disease. Greenhow has pointed out how certain very long standing instances of *phthisis* exhibit an appearance exactly like that of the earlier stages of Addison's disease. Yet the abnormal pigmentation does not deepen or increase, and the symptoms remain only those of the pulmonary malady. Stains on the skin from *pityriasis versicolor* or from *syphilis* have not the characteristic seats of Addison's disease, and they are in patches and surrounded by healthy skin, and certainly the syphilitic affection coexists with other significant eruptions or signs. Malcolm Morris[3] has called attention to the mistake of pronouncing a case of *acanthosis nigricans* one of Addison's disease. The fact that the processes in the former are confined to the upper layer of the skin, and characterized by an abnormal development of the

[1] Crocker, Transact. Clin. Soc. Lond., vol. xiv., 1881; also Carrington, *ibid.*

[2] Medical Times and Gazette, May, 1871.

[3] Medico-Chirurgical Transactions, 1894.

younger not yet cornified elements of the upper layers,—the so-called prickle-layer,—will serve to separate it pathologically from the secondary and relatively unimportant changes of the skin that attend the course of the latter affection. A chocolate-colored discoloration of the whole surface of the body has been observed in a case of psoriasis in which the patient continued to take arsenic during a period of two and a half years.[1] In *Recklinghausen's disease* there is pigmentation of the skin, often in considerable patches, but the subcutaneous tumors due to neuromata, the tumors of the skin of the nature of molluscum fibrosum, the pain, the arthralgia, the alteration of sensation, and the impaired mental activity characterize the affection.

One of the confusing points connected with the diagnosis of Addison's disease is that cases occur without bronzing, or with the discoloration of the skin so slight as to be a matter of doubt. Such cases are generally in persons who die before they have had the disease any length of time. If the altered hue of the skin be wanting, the complaint is undistinguishable from *pernicious anæmia*, except by the characteristic blood-changes this presents. *Other diseases of the suprarenal capsules*, such as cancer and waxy disease, are also not to be separated from the peculiar affection of the gland occasioning Addison's disease, if bronzing of the skin be not present.

The malady, as Greenhow proves, is very rare except in persons employed in manual labor. In some instances it seems to arise from grief or protracted anxiety. The disorder is a chronic one, generally lasting for years; but it almost always destroys life. Yet cases have been recorded in which most of the symptoms of Addison's disease existed and which recovered; and certainly long remissions in the symptoms have been not infrequently observed, and in these remissions the discolored skin has lightened. The disease is occasionally met with in young persons. Dyson reports a fatal case in a girl thirteen years of age.[2]

Pyæmia.—Purulent contamination of the blood is an affection much more likely to be met with by the surgeon than by the physician; yet the physician must be familiar with its symptoms. These are, great depression of the vital powers, high but irregular temperature, profuse sweats, rapid pulse, and the formation of purulent deposits in different portions of the body. The symptoms may be of gradual development; but often they set in suddenly with a chill, to which a fever of low type soon succeeds; or the shivering is followed

[1] Carrier, Medical News, Feb. 3, 1894, p. 127.

[2] Quar. Med. Journ., vol. iii., Part I.

by copious sweating, and the febrile phenomena subsequently appear. A transient erythematous blush on the skin is not unusual.

The pyæmic fever rarely lasts longer than a week, and during its continuance the temperature shows marked variations. Yet the disease is not always alike in this respect; for we find not only cases in which the most decided increase of heat is constantly followed by an equally decided decrease, but also cases in which there are febrile attacks followed by intervals during which the temperature is almost normal. Still, in all the maximum temperature is apt to be very high, ranging from 105° to 108°. Pyæmia, as the physician meets with it, is seen where sinuses or abscesses exist that have no free vent for the pus; or in consequence of an infective phlebitis or arteritis; or in inflammation of the external coat of arteries, with suppuration, especially in the periarteritis of the thoracic aorta; or in ulcerative endocarditis; or the pyæmia results from the purulent breaking down of coagula in the blood-vessels; or it may supervene upon diffuse cellular inflammations, or upon puerperal fever: in fact, it will be found under many dissimilar circumstances. Micro-organisms play an important part in its production, especially the several varieties of the streptococcus pyogenes and the staphylococci. They render the pus septic, and, under conditions favorable to their development, diffuse the process.

There are several complaints with which pyæmia is likely to be confounded, the chief of which are typhoid fever, rheumatism, acute glanders or farcy, and acute affections of the liver.

It is liable to be mistaken for *typhoid fever*, on account of the adynamic character of the fever, and, it may be, the occurrence of diarrhœa and of cerebral symptoms. But the history of the case is very dissimilar: there is no eruption, or, if there be an eruption, it consists, as Bristowe so particularly points out, of sudamina surrounded by a zone of congestion, and is therefore not the eruption of the typh-fevers; and, on the other hand, we find in typhoid fever neither the profuse sweating nor secondary deposits of pus, and the thermometry of the disease is very different. The Widal test would, in any instance, be of value.

The pain in the joints and their swelling in succession, the fever, and the perspirations, resemble much at times *rheumatic fever*. But the difference consists in the greater severity of the constitutional phenomena caused by the poisoned blood, in the marked exhaustion, in the rigors, and in the history not being that of acute rheumatism. Moreover, the frequent signs of formation of abscesses in internal organs or around the joints, the development of pustules on the skin,

and the striking redness of the tumid joints assist materially in the diagnosis.

Acute glanders or *acute farcy* is a disease scarcely distinguishable from pyæmia, since it occasions, for the most part, the same manifestations. The knowledge that the patient who has apparently pyæmic symptoms has been working among horses: the ulceration of the mucous membrane of the nose, and the fetid discharge proceeding from it, which occurs in acute glanders and is apt to be associated with nasal hemorrhages, and with an erysipelatous rash spreading to the cheek and forehead and with enlargement of the lymphatic glands in the vicinity of the affected mucous membrane,—afford us the means of discrimination. Then we find a peculiar tuberculated or pustular eruption, resembling smallpox, upon the skin: and in farcy the lymphatic glands and vessels specially suffer. But most significant are the distinct history of the contagion, the detection of the bacillus mallei in the discharge, and the inoculation test in guinea-pigs producing a characteristic swelling of the testicles followed by suppuration.

Acute affections of the liver resemble pyæmia on account of the jaundice that may attend the latter disorder: the history of the case, the rigors, the sweats, and the purulent deposits distinguish it. Yet it must be remembered that suppurative inflammation of the portal veins and metastatic abscesses of the liver happen, giving rise to pyæmia.

The secondary deposits, or *metastatic* or *embolic abscesses*, take place in the parenchymatous organs, particularly in the lungs and the liver: in the synovial sacs, in muscles, or in areolar tissue, especially in that under the skin. They are mostly due to fragments of septic thrombi that have become centres of suppurative change. If the altered blood coagulate in the arteries, and the infected clot disintegrate, occasioning deposits in solid organs, as in the liver or the spleen, we may have symptoms arising like those of ordinary pyæmia. Indeed, in the *arterial pyæmia*, as it has been called, rigors, febrile symptoms and sweating, and pains in the joints are observable. In connection with the obscure febrile condition, the liver and the spleen are often observed to increase in size slowly.[1] The heart may or may not be affected: ulcerative endocarditis is often present. Hayem has pointed out that there may be capillary embolism in pyæmia, not to be recognized except by the microscope. It may be one of the causes of the so-called *idiopathic pyæmia* in which the source of infection is not apparent.

[1] Samuel Wil[illegible]'s Hospital Reports, vol. xv., 3d Series.

There is a form of pyæmia, called by Leube[1] *spontaneous septico-pyæmia*, which comes on without obvious cause, or is perhaps preceded by a fall or a slight skin wound, in which the symptoms of pyæmia become developed with pain and tenderness in joints and muscles, ecchymosis of the conjunctiva, vesicles in the skin containing blood, extremely high temperature, swelling of the spleen, albuminous urine, pleurisy or perhaps signs of endocarditis or pericarditis, stupor, delirium, cramps, and finally involuntary discharges and coma. The disease, resembling the typh-fevers, or ulcerative endocarditis, is to be distinguished only by the association of the symptoms.

The description of pyæmia given represents it as an acute affection, and so it almost always is. Yet there are cases much slower in their course, and extending over months. These *chronic* or *relapsing* instances of the disease have been described by Paget.[2] The symptoms presented are the same as in the acute disorder; but the local evidences of the complaint are more often seated in different parts of the same tissues, and less frequently in internal organs. The malady is not nearly so perilous as is the acute disease.

Septicæmia.—This is a poisoned state of the blood, produced especially by animal poisons, such as the bites of venomous serpents, or the absorption of putrid matters that have been generated in the economy, or by their inoculation. It may be seen after injuries and wounds, or in the puerperal state. The continued exposure to the breathing of foul air and of septic gases will also occasion septicæmia. There are no discoverable foci of suppuration, but the bacteria occasioning the sepsis are in the main the same as those of pyæmia. Toxines and ptomaines have much to do with the process.

The symptoms of the blood-poisoning vary somewhat with the individual poison that has occasioned it. They are, as a rule, the symptoms of pyæmia, except that secondary pus-formations belong to the former rather than to the latter; and the same may be said of embolism and its results. Rigors are frequently observed. In many instances the altered condition of the blood leads to great prostration, to hemorrhages from internal organs, to petechiæ, to delirium and coma, to extreme rapidity of pulse, to rapidly developed fever with high temperature, to enlargement of the spleen, to cough and bronchial catarrh, and to gastric and intestinal disorders. The blood shows the white corpuscles almost always in marked excess, although not altered in character as they are usually in leukæmia; the red

[1] Archiv für klin. Med., xxii., 1878.

[2] St. Bartholomew's Hospital Reports, vol. i.

globules are diminished.[1] The bacterial types characteristic of the forms of septicæmia are generally demonstrable by microscopic examination and by culture experiments. Staphylococcæmia has been often recognized, and a number of instances of pyocyaneus bacilliæmia have been recorded.[2]

Malarial Septicæmia.—Since the discovery of the *plasmodium malariæ*, malaria has entered the list of infectious diseases, and it has become recognized that it may cause a form of septicæmia. Klebs asserts that he has found flagellate protozoa in the febrile stage of *influenza*, but this observation has not been confirmed.

Typhoid Septicæmia.—Several observers, especially in Italy,[3] have reported cases of typhoid septicæmia without the accustomed alterations in the intestinal tract. The diagnosis was based upon bacteriological examination. Dogliotti also reported a case which had fever, enlarged spleen, and copious eruption of typhoid roseola extending over the entire body. There was profuse diarrhœa. Cultures of blood from the finger and from a vein in the arm remained sterile; but cultures taken from the blood of the papules developed bacilli having all the characters of the typhoid bacilli. At the post-mortem examination no ulcers or cicatrices were found in the intestine. The conclusion is that, besides the familiar form of abdominal typhoid, there is another, identical in every way except that it has no intestinal or lymphatic localization; the bacilli select the skin in preference. Septicæmic typhoid then presents the following characters: an irregular fever, not typical as that of ileo-typhoid; the absence through the entire illness of visceral complications and of symptoms pertaining to the digestive system; the presence in the circulating blood, and especially in the blood extracted from the rash, of a bacillus presenting the characters of the typhoid bacillus.[4]

Pyæmia and septicæmia have shifted greatly from their old significance. We know much more of direct infection and how foci of suppuration are set up in various parts of the body. Another set of symptoms is occasioned when the products of the micro-organisms only, the so-called *toxines*, are absorbed by the blood; here fever, prostration, and various nervous phenomena are caused. This condition has been termed *saprœmia*, to distinguish it from septicæmia, in

[1] Report of the Committee of the Pathological Society of London, Transactions, 1879.

[2] See paper by F[illegible] d Libma[illegible] Journ. Med. Sci., Aug. 1899.

[3] See Guido B[illegible]a

[4] Translation [illegible], vol. xxxviii. p. 203; also Dogliotti, Gazz. Med. di

which the infective agent is actually present in the blood. Occasionally pathogenic micrococci may be present in the body without giving rise to either suppuration or septic disease. The tissue-cells, especially those of the spleen and the kidney, play a very important part in the destruction, and the leucocytes are also active in the warfare.

Thrombosis and Embolism.—Although in connection with endocarditis, with obstruction of the cerebral arteries, and with diseases of the kidney, the phenomena of embolism have been described, it may serve a useful purpose to view here connectedly, though chiefly in their diagnostic bearing, some of the results of the formation of the clots in large vessels or in the heart, and of their being carried along with the current of the blood and driven into remote vessels,—the results, therefore, of thrombosis and of embolism.

In the *veins* thrombi may form, which, so long as they do not produce obstruction of the canal, give rise to no marked signs. A slight hardening and pain on pressure if the coagulum be in one of the more superficial veins, their enlargement if the clot be in a deeper vein, are apt to be the only evidences of the disordered condition. But when the occlusion is considerable, and especially when the collateral circulation is insufficient, œdema is developed, which may be attended with very great tenderness of the swollen part, and, if the impediment be of long duration, with changes in the nutrition of the structures sufficient to produce phlegmonous inflammation. These phenomena are encountered in milk-leg, *phlegmasia alba dolens*, as we see it in puerperal fever, in typhoid fever, in influenza, and in pneumonia; though it is by no means settled whether the thrombosis is primary from the infected blood, or the result of an infective phlebitis. We may have, also, a thrombo-phlebitis as well as a thrombo-arteritis, showing itself as an acute infective fever without anatomical localization, except in the blood-vessels. In some cases profuse hemorrhages happen as a consequence of the stoppage in the vein,—as cerebral hemorrhages produced by *thrombosis of the sinus*, or, as in a case referred to by Virchow,[1] as enormous hemorrhagic infiltration of the subperitoneal and subcutaneous tissues, as well as of portions of the muscles of the abdominal walls, the result of a coagulum in the external iliac vein, the epigastric, and the crural vein. Thrombosis may occur in the cerebral sinuses, without causing hemorrhage, but giving rise to pressure symptoms, pain, prominence of the eyes, and œdema; it may be followed by complete recovery.

[1] Pathologie und Therapie, p. 172.

In *thrombosis of the mesenteric vessels*, the symptoms are intense abdominal pain, great tenderness, vomiting, abdominal distention, and often, if the patient survive the shock, obstruction of the bowels.[1] There is an instance of recovery, the result of an operation.[2]

In *exhausting and wasting diseases*, blood may clot in the veins without any clearly marked cause. Trousseau called attention to the occurrence of milk-leg as a symptom of gastric cancer; and in other kinds of cancer there may be peripheral venous thrombosis; so, too, in Bright's disease. Gout may cause phlebitis and clotting in the veins of the body, as Sir James Paget has pointed out. Undoubtedly infective phlebitis is a cause of thrombosis that is common. But it may be, in a given case, extremely difficult to determine whether the thrombosis or the phlebitis is primary. Again, we may have *chlorosis* give rise to thrombosis in the cavities of the heart and the larger veins, such as the femorals, without phlebitis preceding the morbid condition. The thromboses of chlorosis are generally of the lower extremities, and may be successive and multiple. Both lower extremities are often involved. They are supposed to be due to the feeble circulation of the impoverished blood. But this is not certain. Welsh[3] suggests that the thrombus results from nutritive disturbance of the red corpuscles and their ready disintegration, producing the material leading to a thrombus. The peripheral thrombi in phthisis may be of myotic origin.

Now, portions of the clot, situated in any part of the venous system, may become, by being broken off and driven onward with the circulation, sources of great danger. When coagula occur in the venous system and are wholly or in part carried away with the circulating blood, if we exclude those which, from their situation, could only reach the liver, the manifestations of disturbance arise in the heart or the lungs. Arriving at the *right side of the heart*, the coagulation, if large, or if it become so by serving as a nucleus for a larger clot, occasions symptoms of exhaustion and collapse: an intermitting, feeble pulse: irregular and confused beating of the heart, and cardiac sounds enfeebled or lost over the right side of the organ: rapidly developed distress in breathing, referred, by the sufferer, to the heart, and signs of asphyxia, though all the time the patient is taking deep inspirations: great agitation: and a swollen state of the veins of the

[1] Koester, Deutsche Medicin. Wochenschrift, May 26, 1898.
[2] Gordon, British Medical Journal, June 4, 1898.
[3] Welch, "Thrombosis," Allbutt's System of Medicine, vol. vi., 1897.
[4] R. W. Richardson, Medical Times and Gazette, N. S., 1868.

body. Death may then take place suddenly if a portion of the clot separate and obstruct the pulmonary artery.[1]

But the mode of death, and the symptoms preceding it, in embolism of the *pulmonary artery*, are not always the same, and depend much upon the size of the embolus and where it is arrested. A large-sized clot, whether it be merely part of one occupying the right heart, or be washed at once into the pulmonary artery, will occasion the same signs as those mentioned as indicative of a large clot in the right side of the heart; the craving for air is particularly intense, and this craving is increased by every movement of the body; the muscular debility, the lowered temperature, the cyanosed look, the turgid veins of the neck and their undulations, the increased, irregular cardiac impulse, the disturbed respiration and disordered general circulation, are also noticed; and in some cases a systolic murmur, and, where the case is at all protracted, vertigo, albuminuria, and œdema of the limbs, may be observable. The intellect remains clear. As regards the pulmonary phenomena proper, collapse of the lung, hemorrhagic effusions or so-called infarctions, œdema, or capillary bronchitis are likely to happen, except in those instances in which the principal trunks of the pulmonary artery are blocked up and almost instantaneously asphyxia ensues. If the fragments be very small, the amount of dyspnœa is not great, nor are the symptoms of asphyxia marked; and inflammations of the parenchyma of the lungs may take place, occasioning often secondary obstructions and metastatic abscesses in the lungs, especially when caused by infective emboli.

Blood coagulates in the *arteries* in connection with gangrene, as in diabetic gangrene, and ulceration. Again, inflammation, especially infective, or sclerotic or atheromatous disease of the coats of the arteries may lead to the local development of thrombi; so, it is thought, may feeble action of the heart with increased coagulability of the blood. Arterial thrombosis has much the same symptoms as embolism of the artery, but the interference with the circulation is less sudden and intense, the signs of obstruction change less rapidly, and we often find a cause for the arterial thrombosis in marked arteriosclerosis. Should the case persist, muscular atrophy and trophic disturbance become noticeable.

Still, the most important phenomena connected with obstruction of arteries are those of coagula being washed into them; the phenomena of *embolism*, therefore, rather than those of thrombosis. The manifestations of embolism are distinguished from those of the mere

[1] As in a case recorded by Druitt, Med. Times and Gazette, July, 1862.

formation of clots by what is always the most significant sign of embolism,—the suddenness of the phenomena. And in point of fact the symptoms arise less often as the result of any of the conditions mentioned that occasion coagulation than in consequence of deposits and excrescences which are seated on the valves of the left side of the heart, portions of which deposits are carried away by the circulating blood into remote parts. When these bodies become impacted in a vessel the caliber of which is such that it does not permit them to pass on, we find rapid changes taking place in the portions of the body supplied by the obstructed artery,—coldness, pallor of the parts, a local anæmia, diminished functional activity; and if the first obstruction be followed by others, and the collateral circulation cannot be established, local death and gangrene ensue.

All these changes are, of course, discernible only in external parts, especially in the extremities; the disturbances of function are the most obvious signs where the internal organs are the sufferers. If the emboli be driven to the *brain*, we have often softening as the final result, and this may be preceded by disorder of intellect, without motor disturbances, and by severe attacks of vertigo in cases in which merely the smaller arteries supplying the cerebral cortex are obstructed. But where, as is, indeed, the most common seat of emboli, the arteries of the fissure of Sylvius are clogged, the phenomena are those of apoplectic hemiplegia, and the palsy affects the whole of one side of the body. The brain may also suffer from the seat of the obstruction being in the carotids; indeed, of all organs the effects of embolism are most plainly perceptible in the brain. The presence of emboli in the splenic, renal, hepatic, and mesenteric arteries is generally only to be inferred from the history of the case, and does not occasion any clearly discernible signs. But tenderness, enlargement of the spleen, and pain in the splenic region in *splenic embolism*, or disordered secretion of urine and pain in the loins in *embolism of the renal artery*, or jaundice in *embolism of the vessels of the liver*, may be very marked.

The occurrence of pain in these cases of internal embolism must not be overlooked; and in embolism of the arteries of the extremities pain is a symptom of still greater prominence. It may be like a violent neuralgia, or so constant that it is mistaken for rheumatism; and, as happened in the case of embolism of the right iliac artery, under the charge of Dr. James H. Hutchinson,[1] which I saw with him, it may recur in paroxysms of intense severity, and be referred to the foot, though this be already in a condition of sphacelus. Be-

[1] Amer. Journ. Med. Sci., Oct. 1863.

sides the pain, we find extreme hyperæsthesia in some parts of the affected limb; and pricking sensations, formication, and loss of tactile sense, followed by complete anæsthesia, in others. Then painful spasms of the muscles, and a more or less perfect paralysis of motion, may occur. If we join to these symptoms an absence of pulsation in the arteries below the occlusion until the collateral circulation is decidedly established, a strong beat of the vessel on the cardiac side of the obstruction, the coldness of the limb below this obstruction, and the signs of defective supply of blood, we have a group of phenomena which, taken in connection with the history of the case, render the diagnosis a positive one. In reviewing the history the state of the heart and the cardiac symptoms must be always carefully examined into; and a close inquiry often shows that the sudden manifestations of arterial obstruction were preceded by an attack of palpitation and of irregular action of the heart.

A change in the physical signs of the diseased organ, as of its murmurs, may not be evident; but, should it be evident, it is a sign of utmost moment. Indeed, any change in what may be viewed as the centre from which the embolus may be detached is of great significance. And this holds good quite as much for venous as for arterial emboli. Thus, in a case of coagulum in a vein, a sudden disappearing of swelling and œdema of the affected limb, with the supervention of signs of embarrassed circulation and respiration, would at once tell what had taken place.

In regard also to the diagnosis of embolism we must always bear in mind the causes that are likely to give rise to it. Several of the causes of arterial embolism have been already mentioned; those of venous embolism are the same as of venous thrombosis, or, to speak more explicitly, the breaking up of the clots and their transportation may occur in any of the conditions which have occasioned them. Now, these conditions, too, will produce arterial clots, and indeed some are more apt to lead to coagulation in the arteries than in the veins. Prominent among them are a narrowing of the caliber of the vessel, as by pressure; dilatation of the vessels and of the heart; failure of cardiac power, with consequent retardation of the blood-stream, —a state which is more likely to occasion venous than arterial thrombosis; a breakage in the continuity of the vessel, as when it is torn or cut; changes which take place in the coats of the vessels, especially inflammatory changes, the result of infective disease; and contact of the blood within the vessels with foreign bodies. Then it is very likely that special states of the blood, by altering the cohesion of the globules, or disintegrating them, may cause the clotting.

Another cause of embolism is that due to *accumulations of pigment in the blood*, the result of malarial fever. The pigment may obstruct the capillaries in the brain and thus occasion capillary apoplexies; or be driven to the liver and there produce signs of disturbance of its circulation, and abscesses. As in all forms of capillary embolism, the symptoms are obscure: the suddenness of their development, generally so characteristic of the other forms of embolism, is wanting; and the diagnosis, as throughout in capillary embolia, is nothing more than a matter of conjecture, based on a close study of the history of the case and the general phenomena, including the microscopic examination of the blood. Similar symptoms occurring after fractures of bone point to emboli derived from the marrow, to *fat embolism*.

Acute endarteritis may be the cause of embolism as well as of pyæmia. *Air in the blood* produces great disturbance of the circulation, which may be thought to be due to embolism. The air may be the result of decomposition, and get into the venous system and thence into the general circulation. Jurgensen[1] has reported a case in which the air passed into the circulation through the splenic vein. Irregular contraction of the heart, pallor of the face, a peculiar systolic cardiac murmur, faintness and the signs of cerebral anæmia, and slow breathing, are the common symptoms.

In conclusion, the *subsequent changes of the thrombus* must be adverted to. It may organize and be converted into connective tissue and yield an impaired passage to the blood; and perhaps the collateral circulation may be freely established; or, what is not so favorable a result, it may soften and undergo a granular and fatty degeneration. Further, septic or purulent thrombi, as they soften, may produce septicæmia or pyæmia, or particles of the thrombus may be wafted into capillaries and there lay the foundation of abscesses. It is thus that in a case of thrombus or embolus we may have the results of a secondary pyæmia to deal with.

Scurvy.—This disease is not often met with in civil practice; but it is one familiar to the military and the naval surgeon. It consists in a deterioration of the blood, produced by living for a long period upon the same kind of food, especially upon salted meats, without the requisite supply of fresh vegetables. Another cause of scurvy is the want of proper assimilation of food, as in prison scurvy.[2]

The existence of scurvy in childhood is now recognized as of not infrequent occurrence, and it is probably frequently mistaken for

[1] Archiv f. klin. Med., Bd. xxxi., 1882.

[2] See Medical Memoirs of the U. S. Sanitary Commission, p. 278.

rhachitis, acute rheumatism, or possibly for purpura. The concurrence of marked anæmia with joint-swellings in a bottle-fed infant, or in older children, should suggest the possibility of scurvy being present. Northrup and Crandall[1] found, in over sixty-three per cent. of the cases of infantile scurvy they investigated, that the diet consisted of proprietary foods and condensed milk. The evidence also indicates that milk sterilized is capable of causing scurvy when used as an exclusive diet.

Babes,[2] in studying three cases of scurvy bacteriologically, found a thin, long, wavy bacillus, prone to occur in clusters, in the gums, the lungs, and other viscera. There were also streptococci in the gums. The blood shows nothing characteristic. The red corpuscles are only slightly diminished, the hæmoglobin more decidedly, giving, as Coles[3] says, a chlorotic type to the anæmia.

Scurvy is usually slow in its development. The patient becomes low-spirited, easily fatigued, and is loath to exert himself. The appetite is impaired; there is a craving for acids and for vegetable food; the tongue is flabby; the breath fetid; the pulse feeble; the skin dry. The bowels are usually constipated; but a tendency to diarrhœa may exist, and indeed generally occurs as the disease advances. Neuralgic pains, referred chiefly to the lower extremities, to the bones, and to the back or thorax, are common. The face is pale, or has a yellowish tinge; the eyes are surrounded by a dark ring. During the progress of the ailment, or in severe cases almost from the onset, we find swollen, spongy gums, bleeding on the slightest touch; hurried breathing; a rapid pulse; weakened eyesight, sometimes night-blindness; epistaxis; painful swelling and hardness about the joints of the extremities and in the calves of the legs; and purple spots and bruise-like stains on the skin. Should the malady remain unchecked, the symptoms heighten in severity, ulcers form which have a fungoid look and a great tendency to bleed, hemorrhages take place from internal organs, old sores and wounds reopen, well-knit fractures become disunited, there is a constant tendency to swoon, and the patient perishes miserably exhausted, and with his blood in a state of dissolution. Scurvy may be the cause of epidemics of pericarditis.[4] In some cases death takes place from diarrhœa or dropsy, which may be suddenly developed. Recovery from scurvy is slow.

Purpura.—Scurvy is not a disease difficult to recognize; only one

[1] Proceedings of New York Academy of Medicine, Feb. 1894.

[2] Quoted in Sajous's Annual, vol. i., 1895.

[3] Diseases of the Blood.

[4] Von Dusch, Herzkrankheiten.

affection resembles it at all closely,—*purpura*. In this disorder also red or purple spots or livid blotches, uninfluenced by pressure, and passive hemorrhages from the mucous membranes happen. But there is this difference between the two complaints: purpura is common in fruit seasons, and often attacks persons who have not been in any way deprived of vegetable food. The gums are not soft and spongy as in scurvy, nor do we find the same weakness of mind and body. Then, the stain of the skin in purpura is apt to be more generally diffused, and the purple blotches are smaller, or, at all events, the large patches of discoloration consist clearly of an aggregation of very many small spots. Moreover, the disorder is not controlled, like scurvy, by antiscorbutics, such as fresh vegetables and lemon-juice.

From a clinical point of view we find several forms of purpura. In the mildest, the purpurous spots are apt to appear only on the legs. They come in crops, which fade, and there are no constitutional symptoms, except a little lassitude, and perhaps aching of the limbs and pain in the back. In the graver cases, "purpura hemorrhagica," we have, in addition to the cutaneous hemorrhage, epistaxis, hæmatemesis, hæmaturia, or other internal hemorrhages, and extravasations of blood may happen into the substance of the muscles. The amount of pain attending the malady is very different. There may be none, or it may be trifling: or deep-seated pains in the cavities of the body, or extended neuralgic pains, may accompany the purpurous complaint; there is at times soreness at the points of extravasation. In some instances the pains are chiefly felt in and around the joints, and the apparently rheumatic aches subside in a few days, and spots of extravasated blood become visible. This "purpura rheumatica" is usually met with in the strong and healthy. It is, indeed, one of the peculiarities of any kind of purpura, that it may come on in the midst of seemingly excellent health: for while it is true that the disorder may be preceded by general debility, or occur in the course of disease of the liver, of Bright's disease, or as a sequel to the exanthemata and rheumatic fever, it most often happens where we should least expect it. Its production as the result of a sudden shock, such as fright, and its intermittent character, have been repeatedly noticed. It has appeared after the administration of quinine, as observed by Vépau,[1] by Gauchet,[2] and by Woodbury.[3]

The blood shows nothing peculiar, nothing but the form of anæmia common after hemorrhages,—reduction of the corpuscles and ha-m-

[1] Gazette Méd. de Strasbourg, 1865.

[2] Bulletin de Thérapeutique, vol. el.

[3] Philadelphia Medical Times, 1886.

globin, slight increase of the white, occasionally nucleated red cells. Purpura is clearly not merely a disease of the blood; the capillaries lose their retentiveness and allow the corpuscles to migrate. The duration of the malady is very variable: only a week may elapse, or several months may pass, before the spots disappear. In some cases purpura presents an *acute* form. It is ushered in by a chill, and by intense pain in the back and limbs, but is unattended with fever or severe constitutional disturbance. The purple spots usually first appear on the legs, and are wholly uninfluenced by pressure. They last five or six days, or somewhat longer, then gradually change their color and fade. Even in marked hemorrhagic cases, the mind remains clear, and cerebral or spinal symptoms are absent. It is thus that we are able to distinguish severe cases of acute purpura, which may indeed prove fatal in forty-eight hours,[1] from cerebro-spinal meningitis. Some of these acute or fulminating cases occur in young children, and it is a question whether or not they were in reality subjects of infantile scurvy.

The distinction between *hæmophilia* and purpura is simple. It is true that in the former the bleeding may happen into the skin, or from any of the parts from which it may take place in purpura; but the family history, the congenital proneness to frequent hemorrhages from the slightest cause, their danger and protraction, the functional disorder of the heart, followed perhaps even by cardiac hypertrophy, the attacks of rheumatoid joint-inflammation, especially after exposure to cold and damp, and the hemorrhagic diathesis exhibited, stamp hæmophilia with distinctive features.

Microscopic examination of the blood of hæmophilia shows nothing different from the anæmia found in scurvy or in purpura. Microcytes, and reduction of hæmoglobin and of the red blood-corpuscles, were found by Daland.[2] Henry has directed attention to the wasting of the middle muscular tunic of the arterioles. Vasomotor influences undoubtedly play their part in bringing about an attack. This is shown by the flushing of the face which so often precedes it, and also by the fact that the attack may follow emotional excitement. *Leukæmia* may be accompanied by subcutaneous extravasation of blood, but it cannot be mistaken for either hæmophilia or purpura, if an examination of the blood be made. Hæmophilia is almost exclusively restricted to the male sex.

[1] Harrison Allen, Amer. Journ. Med. Sci., Jan. 1865.

[2] College of Physicians of Phila., Jan. 9, 1894.

CHAPTER XI.

RHEUMATISM AND GOUT.

RHEUMATISM AND GOUT are affections having a strong tendency to change their seat, and are dependent upon the presence in the blood of some poisonous material which probably accumulates there in consequence of malassimilation. The rheumatic poison, concerning which there is a growing but as yet unproved belief that it is a micro-organism, has a singular predilection for the fibrous, serous, and muscular textures. Hence we find it attacking principally the joints, the fasciæ, the endocardium and pericardium, and the muscles in various parts of the body. According to its main forms, it is sometimes divided into articular and muscular; but the more usual division into acute and chronic is simpler.

Acute Rheumatism.—Here the poison gives rise to the symptoms of an acute, active disease, and attacks especially the larger joints. These swell, become hot, red, tense, tender, and the seat of pain, aggravated by the slightest movement; an effusion also takes place into the surrounding structures, or into the synovial membranes of the joint itself. The rheumatic inflammation may either remain confined to the joints first affected until the disease is over, or, what is more common, it shifts from joint to joint, implicating most of the large ones in succession, yet often invading fresh joints before the swelling has subsided in the parts first attacked. The articular disorder is ushered in and accompanied by high fever, soon attended with a full, bounding pulse, profuse, sour perspirations, a white, coated tongue, and a scanty, turbid, highly acid urine. The fever is generally in proportion to the number of joints involved. Tonsillitis is not an unusual early symptom. The temperature runs up to about 102° or 103° F. very soon after the outbreak of the malady, and remains steady, with slight evening exacerbations and morning remissions when the joint-affection is yielding, but with renewed rises when fresh joints are being implicated. As the disease disappears, the fever temperature gradually subsides.

There is little difficulty in recognizing the complaint. The pains in the joints, their tumefaction and tenderness, the shifting character

of the disorder, the fever, the acid sweats, form a group of phenomena eminently characteristic. In truth, excluding acute gout, the only affections at all likely to be confounded with acute articular rheumatism are pyæmia and glanders, acute synovitis, and milk-leg. The diagnosis of the former has been discussed in connection with diseases of the blood; it only remains to point out the marks of similitude and contrast between acute articular rheumatism and the other maladies.

Acute synovitis resulting from an injury, or from cold, occasions, like articular rheumatism, pain and heat in the joint, with distention. But the disorder, except, perhaps, if it happen in a rheumatic constitution, does not affect more than one joint; and, as there is scarcely any, or no, effusion into the surrounding tissues, the outline of the joint can be distinctly discerned, and fluctuation is readily detected. Often, too, the accumulation of fluid reaches an extent far greater than in rheumatic inflammation; moreover, the febrile and constitutional derangement is not so severe as in acute rheumatism, and the affection has no tendency to change its seat. Still, acute synovitis may be rheumatic.

Milk-leg, or phlegmasia alba dolens, occurs usually in women after delivery or as a sequel of continued fevers, sometimes in pneumonia, in chlorosis, or in tuberculosis. Generally, only one leg swells, and this becomes throughout, or only around the calf, preternaturally white, firm, hot, and shining. The tumefaction is uniform, and painful, especially so when touched. It does not pit, or pits but slightly, upon pressure, except at the lower part. There is tenderness with a sense of hardness in the crural, the femoral, or the internal saphenous vein, though this is by no means constant; yet phlebitis or periphlebitis of infective origin, whether primary or secondary, is apt to be present, and to be associated with a thrombus in the vein. The history of the case and the local signs are very different from acute rheumatism. Among the latter, two giving rise to striking dissimilarity may be mentioned: the almost entire loss of power in the affected limb in phlegmasia alba dolens, and the much higher temperature it shows by the thermometer than the other members. An increase of general temperature corresponds to an increase of pain and swelling in the limb, and of constitutional distress.[1]

Rheumatism may be modified in its manifestations by happening in connection with, or consequent upon, other disorders. For instance, the febrile phenomena may be of an adynamic type when the disease occurs consecutively to typhoid or typhus fever. In the

[1] Elliott Richardson, Pennsylvania Hospital Reports, vol. ii.

course of certain infective diseases, such as scarlet fever, dysentery, cerebro-spinal fever, and gonorrhœa, or in septic states, such as in pyæmia, the joints swell, and there may be symptoms like those of rheumatism.

In *gonorrhœal rheumatism* the articular pain is not so severe or acute; the integument covering the affected joint is apt to retain its normal color; there may be but one joint—and there are not generally many—implicated; the inflammation is confined to the synovial membrane, and a copious sero-fibrinous exudation occurs; the joint-affection, which is found chiefly in the knees or the sacro-iliac or the sterno-clavicular joints, shows a slight tendency to shift, and resembles rather an acute or a subacute rheumatoid arthritis than acute rheumatism; the eye, too, unlike what happens in ordinary acute rheumatic fever, is often attacked. There is but little fever, no copious sweating, and no disturbance of the heart, though there may be, in rare instances, a coexisting gonorrhœal endocarditis; often there has been a discharge from the urethra, which diminishes when the gonorrhœal rheumatism sets in, but which does not cease. The disorder does not come on early in a case of gonorrhœa; and the joint-affection appears really to be of pyæmic origin. It disappears only very slowly, and is uninfluenced by salicylic acid.[1] It is rare in women. In two hundred and fifty-two cases analyzed by Northrup there were only twenty-two women. Gonorrhœal rheumatism may run an acute course.[2]

Purulent effusions into joints may be mistaken for acute rheumatism. The history of the case, the frequent association with an infectious or septic malady, and the location of the swelling, distinguish these *pyæmic joints*. They are also met with in puerperal fever. In *acute osteomyelitis* happening in the long bones near the joints there may be misleading symptoms. But the great severity of the pain, the fact that the epiphyses rather than the joints are affected, and the grave constitutional symptoms prevent error.

The traits of an attack of acute rheumatism are frequently altered by certain complications in internal organs which the contaminated blood occasions. Prominent among them are the cardiac disorders, which are in fact so common that they may be looked upon as forming part of the rheumatic manifestations. Their signs we investigated while examining endocarditis and pericarditis. Certain cardiac phenomena, such as extreme pain without evidence of valvular affection,

[1] German edition of this work.

[2] Davies-Colley, Guy's Hospital Reports, 1883.

pain which may shoot to the neck and shoulder and be associated with signs of great irritability of the heart or of heart-failure, have been by Peter and Letulle[1] attributed either to rheumatic myocarditis, or to an abnormal excitement of the cardiac plexus, of rheumatic origin.

Other complications are inflammations of the lung, of the bronchial tubes, and particularly of the pleura; an affection of the kidney which is generally a parenchymatous nephritis with some albumin and tube-casts, but which may be due to pyæmic or embolic infarction,[2] and—though not often—cerebro-spinal disturbances, exhibiting themselves by headache, violent delirium, convulsions and coma, and occurring either in connection with peri- or endocarditis, or solely in consequence of the action of the vitiated blood on the nervous centres, or of uræmia, or of multiple capillary embolism. In these cerebral cases the temperature is apt to be very high, to reach 107° or more, but the association is not invariable. Indeed, rheumatic delirium is far from always of the same nature. It may develop itself with or without the signs of cardiac complaint. It may come on early in the disorder during the violence of the fever; or late, and clearly from debility and impoverished blood, yielding to nourishment and stimulants. It is rarely the result of meningitis. The delirium which attends *cerebral rheumatism* may be marked by great talkativeness, or, on the other hand, the patient may be extremely taciturn.[3] Insanity may follow the brain symptoms of acute rheumatism. In some instances, whether due to rheumatic inflammation or to mere disturbance of the medulla and lower half of the pons, we find in rheumatic hyperpyrexia nervous symptoms that simulate multiple sclerosis,—exaggerated knee-jerks, ankle-clonus, scanning speech, nystagmus, and tremor. Foxwell[4] has reported such a case in which the temperature reached 111°.

The occurrence of *nodules* in connection with rheumatism, especially among children, has attracted much attention. They are met with chiefly in the neighborhood of joints, especially of the elbow. These fibrous nodules may appear at once in any form of rheumatism, or come out in crops. They are not tender. They most often occur in cases of rheumatic endocarditis or pericarditis.

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[1] Archives Générales de Médecine, June, 1880.

[2] Chomel, Recherches sur les Reins dans le Rhumatisme, Paris, 1868; also Schmidt's Jahrb., No. 2, 1871.

[3] Some of these points are more fully detailed in my paper on Cerebral Rheumatism published in the Amer. Journ. Med. Sci., Jan. 1875.

[4] Lancet, May 1886.

In a few instances of rheumatism we find *acute arteritis* arising, and especially inflammation of the fibrous structures of the aorta. This condition may be suspected should we observe intense general uneasiness and distress, with pain, increased pulsation, a distinct murmur in the course of the vessel, and tumultuous action of the heart without there being obvious signs of disease of that organ present. Still, the diagnosis is never a positive one.

Acute rheumatism rarely ends fatally; its cardiac consequences are more to be feared than the acute attack. Cases occur not infrequently in which the inflammation in the joints is lingering, and in which the febrile symptoms are not intense. These cases form an intermediate grade between acute and chronic rheumatism, and are spoken of as *subacute*. The disorder is more apt than the acute variety to affect the muscles as well as the joints; nay, the former may be alone attacked. It may be witnessed in the joints of one extremity, or in one joint, and might then be mistaken for synovitis. But the dissimilar history of the complaint will guard against error: no accident has happened to account for the swelling of the joint, and often the patient will tell us that he has had previously an attack of rheumatism. The subacute form of rheumatism is more likely to be confounded with rheumatic arthritis: we shall presently refer to their distinction.

Chronic Rheumatism.—This may be either a sequel of the acute disease, or the disorder may from the onset assume a lingering form, the constitutional symptoms being slight. The affection may show itself in the joints, giving rise to stiffness, dull aching, pain produced by motion, but without heat or obvious swelling, tenderness, and febrile excitement, or marked sweating; or it may implicate the muscles in various parts of the body, occasioning stiffness, as well as pain when they are moved; or it may attack both joints and muscles. In any case the occurrence of the pain furnishes the starting-point in diagnosis; and we must ascertain whether it be augmented by motion, whether it be more or less shifting, whether it be not combined with stiffness either of the muscles or of the joints, whether it be influenced by changes of temperature, whether it be not neuralgic, or associated with a disturbance of some viscus, such as of the liver or the kidneys, before we conclude that the complaint is really rheumatic.

This is especially necessary in the most common form of chronic rheumatism,—*muscular rheumatism*. All kinds of pains in the muscles or their surroundings, the cause of which is not at once apparent, are apt to be pronounced rheumatic. And indeed it is not always easy to say whether they are or are not of that character. We may

distinguish them from *neuralgia* by the pain in the latter being ordinarily confined to the distribution of one nerve and not being increased by movement or by pressure, nor is it so steady, or attended with soreness, except over a few spots in the course of the affected nerve, which then, indeed, bespeaks neuritis rather than neuralgia.

As regards the pain caused by *organic structural disease*, we can generally discriminate them from those of rheumatism by close attention to the history of the case, and by a careful exploration of the internal organs. Thus, for instance, we shall find pain radiating from the right hypochondrium to the shoulder to be dependent upon hepatic disease; or pain shooting down to the groin, thigh, and testicle to be caused by a disturbance of the kidney; or a bearing down and an aching near the sacrum to be probably due to uterine disorder, prostatic disease, or anal fissure.

Muscular rheumatism may affect the neck, the scalp, the muscles of the face, and the parietes of the chest or of the abdomen. It may be not only chronic in any of these situations, but also acute; or what is more frequent, when it occurs with fever and is transient, it is a sudden acute exacerbation in persons who are rheumatic and suffer more or less persistently from rheumatism, though perhaps in a different part of the body from the one in which the acute affection has happened. Muscular rheumatism has been noticed in an epidemic form.[1]

One of the most common seats of muscular rheumatism is in the loins. It then constitutes the disease known as *lumbago*. The patient is unable to stand erect, or, after being seated, to assume the erect posture without suffering much pain, and finds it nearly impossible to stoop forward, on account of the pain occasioned when the muscles of the back are called into action. Unless the attack be very severe or acute, there is no constitutional disturbance; but the disorder is often obstinate. We distinguish it from pain in the loins due to disease of the kidneys, chiefly by an examination of the urine and by the way in which movement affects the rheumatic pain; from lumbo-abdominal neuralgia, by the two or three sore spots in the course of the affected nerve; from rheumatism of the vertebral articulations, by the absence of tenderness and swelling around the spinous processes; from lumbar abscess, by the want of local bulging or fulness, of fluctuation, and of fever. Then, we must be careful not to consider as lumbago the pain in the back caused by disease of the spine, or by disorder of the uterus, or by the passage of abnormal

[1] Schmidt's Jahrb., No. 12, 1872.

urinary constituents, such as oxalate of lime, or consequent upon strains, or blows, or scurvy, or malaria, or anæmia, or a general or local muscular debility.

Thus there are many causes of pain in the loins, and where the case is of any duration or of any doubt we must be careful to exclude these causes from consideration before we assume the disease to be really rheumatism of the muscles and fasciæ of the back. This caution is very necessary in investigating the cases of "weak back" so prevalent among soldiers, which are, for the most part, due to strains or injuries that have perhaps produced a weakness of the muscles and a persistent cutaneous hyperæsthesia; or to impoverished blood, to neuralgia, to scurvy; or to digestive disorders attended with the passage from the kidneys of large amounts of urates or of oxalate of lime.

The remarks made with reference to lumbago and the states which simulate it are also applicable to pains apparently muscular affecting other portions of the body. We may have pain and soreness of the muscles developed by strain or overwork and attended both with muscular and with cutaneous hyperæsthesia,—a condition very different from rheumatism, and designated *myalgia*. This soreness of the muscles is always in direct proportion to their debility, and is chiefly caused by long-continued exertion beyond the power of the muscle, or by an ordinary amount of action when the muscle or the individual himself is debilitated. The morbid state is very marked during convalescence from scarlet fever, where it may be looked upon as due to over-exertion of the weakened muscles. The soreness of the muscle is commonly accompanied by heightened sensibility of the skin over it; and this coexisting cutaneous tenderness is an important diagnostic sign. Myalgia is chiefly found in the muscles of the trunk, and is rarely general.

Another form of muscular involvement that we may here mention is wry-neck, or *torticollis*. This depends chiefly upon contraction of the sterno-cleido-mastoid muscle of one side, and occasions an ungainly appearance. But every case is not of rheumatic origin. The disorder may be spastic, or may depend upon nervous injury, and when chronic may lead to alteration in the muscular structure. The therapeutic test is with injections of atropine, hypodermically, which are generally useful, not only for their remedial effect, but also because, even in chronic cases, they may show us, by the difficulty or impossibility of relaxing the muscle, how much of it is really changed.

There are forms of pain in muscles and tendons that are often mistaken for muscular rheumatism. *Achillodynia* is one: the pain

swelling about the insertion of the tendo Achillis, with pain on standing or walking but without much tenderness, marks an affection that is frequently not rheumatic. In *Morton's disease* the pain in the metatarsal phalangeal articulation of the fourth toe is due to nerve-compression. It is a form of neuralgia, which occurs in seizures, yet only when the foot is moved as in walking; there is neither heat nor swelling. The muscular pains of *trichiniasis* may be mistaken for muscular rheumatism. But the marked exhaustion, the signs of gastro-intestinal catarrh, and an examination of the blood direct attention to the real cause.

A form of chronic rheumatism which also may be briefly mentioned is that affecting chiefly the fibrous membranes, such as the *periosteum*. This becomes thick, and tender on pressure; its thickening may be even perceptible to the touch as well as to the eye. This kind of rheumatism happens especially in those who have syphilis; but it also occurs where no such taint exists. The pains are generally much more severe at night; and this is sometimes assumed to be a proof of the syphilitic character of the disease,—but incorrectly so; for many varieties of chronic rheumatism are aggravated by the warmth of bed. Indeed, the only really diagnostic signs of syphilitic rheumatism are the obvious evidences of constitutional syphilis, or the history of the infection. Still, to cases in which several nodes exist, and in which the pains more particularly affect the long and flat bones, and in which iodide of potassium speedily modifies the pains, we shall be rarely wrong in attributing a syphilitic origin.

Chronic rheumatism is often feigned, especially by malingerers in the army and the navy, and the deception may be difficult of detection. They pretend to be scarcely able to walk, or hobble around with a cane, and complain much of the pain and stiffness in their joints. Yet there is not the least sign of deformity or real stiffness; the pain is always stated to be the same; and their general health is excellent. Their way of using the stick, too, is characteristic: they move it each time they move the seemingly crippled leg, but, as a rule, not immediately, thus not employing it as a support. Anæsthetics are of great value in enabling us to decide as to the real amount of immovability of the limb.

Gout.—This disease may be, like rheumatism, either acute or chronic. Instead of describing its phenomena, I shall at once point out the marks of difference between the two kindred maladies. In gout, the small joints are chiefly or alone affected; in rheumatism, the large. The gouty inflammation is accompanied by more local pain and redness than the rheumatic, and by œdema, enlargement of

the veins, and desquamation of the cuticle, and implicates, at least at first, only one or a few joints, especially the joint of the great toe; while rheumatism attacks the joints of the upper as well as of the lower extremities. In gout there is a tendency to disease of the kidneys, with a moderate febrile disturbance, and no profuse sweats; but we meet rarely with a cardiac complication, at least a valve affection, as constantly happens in rheumatism. Gout is more decidedly hereditary than rheumatism; its early attacks are apt to recur with a certain amount of periodicity, and last about a week,—therefore a much shorter time than those of rheumatic fever. During the paroxysm of gout the urine is scanty, and both before the attacks and during the first days the uric acid is strikingly diminished.

Gout occurs generally in men of middle age who live high or who drink large quantities of malt liquor, or in their descendants, particularly those who lead inactive lives; it also is seen in those whose systems have been impregnated with lead; while rheumatism is usually met with in the weak, is excited by cold and damp, is almost as common in females as in males, and is oftener found in the young and before middle age. Gout is frequently combined with a deposition of chalk-stones in the joints; rheumatism never. Then, as shown by Garrod,[1] we possess means of diagnosis in the examination of the blood. Uric acid is always present in large excess in gout, and absent in rheumatism. Nor is the method of detecting the uric acid difficult, if we make use of Garrod's ingenious plan. It consists in obtaining the crystals of uric acid, crystallized on a thread placed in a mixture of the serum of the blood, or of the fluid from a blister, with acetic acid, in the proportion of six minims of the acid to each fluidrachm of the serum. The mixture of the serum and acid, with the thread in it, is allowed to stand in a shallow watch-glass from twenty-four to forty-eight hours, protected from the dust. In the blood of gouty patients there is often a slight increase of the leucocytes.

The remarks just made apply more especially to the distinction between acute gout and acute rheumatism. The chronic disorders are more difficult to separate. Indeed, unless there be external deposits or chalk-stones, their discrimination may be impossible. In these obscure cases, however, the history and an examination of the blood may throw considerable light on the diagnosis. In many subjects, too, the exploration of the external ear will assist us in arriving at a correct diagnosis: we find one or several spots of subcutaneous deposit of urate of sodium on the helix.

[1] Gout and Rheumatic Gout, 2d edit., London, 1863.

Gouty persons are subject to indigestion, flatulency, pains and cramps, or palpitation of the heart,—phenomena due to the gouty poison, and generally ameliorated by a fit of gout. The teeth of those of gouty diathesis are remarkably well enamelled, enduring, and free from decay; but there is great proneness for tartar to collect upon them.[1] Violent fits of sneezing may be a most annoying symptom,[2] and so are deep-seated pain in the tongue and a sense of burning.[3] In chronic gout there are often knotty finger-joints and tophaceous deposits in fingers and toes. Gouty endarteritis is not uncommon; and the frequent association of contracted kidney with gout is universally recognized. Hay fever, or asthmatic seizures, may be symptomatic of the gouty diathesis or lithæmia.

The gouty inflammation of the joints may retrocede during an attack, and severe epigastric pain, nausea, vomiting, flatulence and acidity, faintness and a feeling of sinking, and a quick, feeble pulse show that the morbid action is transferred to the *stomach;* or it flies to the *head*, and apoplexy or maniacal symptoms occur; or to the *heart*, and there is violent palpitation, with dyspnœa and intense anxiety; or it attacks the *spinal cord*, and a sense of constriction around the thorax and abdomen, and piercing pains in the limbs, like those of locomotor ataxia, are encountered, and the spinal dura mater and the roots of the spinal nerves are found to be incrusted with uric acid and urate of sodium.[4]

Closely connected with gout is *lithæmia*. Indeed, the excessive formation of lithates and the dyspeptic symptoms with heart-burn and eructations, the signs of functional derangement of the liver, the vertigo, the mental gloom or the listlessness and indisposition to exertion, the cramps in the legs and muscular twitchings, the neuralgic attacks, the restless nights, the palpitations of the heart and its irregular beat, are in many but the precursors, although, it may be, the long precursors, of a regular outbreak of gout; while in many more this half-dyspeptic, half-nervous condition, with the faulty assimilation, the imperfect oxidation, the excessive discharge of lithates at times and their disappearance at other times, will go on for years without ever developing into an attack of gout.[5] Still, in time, the same local lesions may follow in internal organs; we may have

[1] Dyce Duckworth, Transact. Odontol. Soc. of Great Britain, 1883.

[2] Schmidt's Jahrbücher, No, 8, 1881.

[3] Dyce Duckworth on Gout, London, 1889, p. 87.

[4] Ollivier, Archives de Physiologie, 1878.

[5] See paper on Lithæmia, by the author, Amer. Journ. Med. Sci., Oct. 1881; and University Medical Magazine, May, 1894.

the same form of contracting kidney, arteriosclerosis, and the heart-affection with hypertrophy, and the accentuated second aortic sound of the lithæmic state. Lithæmia is very common in this country, and may be termed American gout.

Lithæmia sometimes manifests itself in attacks of pain in the *stomach and bowels*. The pain is associated with tenderness, and is most common when the stomach is empty. Among the symptoms of lithæmia that are very liable to be mistaken and mistreated are *disorders of vision*. As Risley[1] has stated, lithæmia is both a primary and a modifying factor in many of the discomforts and more serious disorders of the eye. It stands second only to syphilis in the frequency with which it causes iritis. In adults, obstinate conjunctivitis and episcleritis are apt to own lithæmia as a cause, and it often gives rise to pain and to photophobia. It may lead to ulceration of the cornea and errors of refraction and attendant eye-strain and headache.

Arthritis Deformans.—Gout is rare in this country. But the same cannot be said of that distressing disorder known as *arthritis deformans*, or *rheumatic gout*, which is neither rheumatism nor gout, —though not uncommon in those of gouty history,—but a distinct affection. The disorder may be acute or chronic. It is not often the former; many of the acute cases, indeed, being rather subacute than acute. Even in those belonging to the *acute* form there is little febrile disturbance; and though we observe pain and aching in the joints, and some discoloration, we find less redness than in acute rheumatism, and certainly the tongue less furred, much less profuse perspiration, no such heavy deposits in the urine, and an utter freedom from cardiac complication. The acute arthritic disease has rather inflammation of the pleura and of the eye as its attendants, and is often accompanied by a sallow skin, yellowish conjunctiva, and discolored, costive stools. It implicates the large and small joints equally, thus differing from gout, and causes very great swelling, due to an effusion, not around the joint, but into its capsule. It fastens upon several joints, and, though it may pass from joint to joint, it shows but little migratory tendency; the joints first attacked remain the seat of disease. Unlike gout, it is apt to affect the smaller joints of the hands without a previous affection of the toes, and exhibits no periodic paroxysms or exacerbations. Moreover, an acute attack is of very much longer duration. Unlike subacute rheumatism, it does not affect the muscles, and is, both in the suffering at the time and in its ultimate results, a much graver malady.

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[1] Proceedings of the State Medical Society of Pennsylvania, 1895.

The great danger in deforming arthritis is from the effects of the inflammation on the joints. The changes there produced are obvious in the *chronic* form, for each joint attacked is apt to be much damaged. The chronic complaint may follow the acute, or it may begin without any febrile symptoms, with pain and stiffness in the joints. These soon become much distended with fluid, which is gradually absorbed, and the structure of the joint alters, the cartilages become, sooner or later, implicated, and gradually waste, and chronic changes and permanent deformity are produced. The alterations may go on getting worse and worse in consequence of repeated attacks, until complete immobility ensues, and, the joints becoming permanently affected, the ends of the bones are dislocated and enlarged. But though there is much swelling, no deposits of urate of sodium are found in the joints. The appearance of the joints seen with the X-rays is very characteristic. The enlargement and irregularity of the articulating surfaces and the bony outgrowths at the margins are conclusive evidence of the affection, and unlike anything perceived in either rheumatism or gout. Occasionally, especially in men, the disease is only found on one side of the body, and may show itself only in a large joint, as in the hip or the shoulder, or affect only the spinal column, producing immobility. Among its peculiar, though less constant symptoms, are very rapid pulse, sweating, and pigmentation of the skin, like freckles. In one of the forms of the disease, little nodes are found, especially at the sides of the second phalanx of the fingers, and gradually increase in size. These "Heberden nodes" in time become associated with eburnation of the ends of the bones.

Charcot has pointed out that in *paralysis agitans*, in addition to rigidity of the muscles, deformities of the fingers result resembling closely those of chronic articular rheumatism. But the likeness to the deformities caused by rheumatic gout is still closer, and to distinguish them we must take into account the whole history of the case, the tremor, the fixed look, the peculiar gait, the indistinct speech, the tremulous handwriting, the sensation of excessive heat. Moreover, the disfigured joints are not stiff, and do not crack. The *arthropathies* of *locomotor ataxia* may be mistaken for arthritis deformans, but, irrespective of the history and of the characteristic pains, the absence of the patellar tendon reflex distinguishes them. All these joint affections following nervous diseases, and sometimes classed together as *spurious arthritis*, differ from joints attacked by rheumatism or by deforming arthritis in the absence of marked swelling and of pain, except on forcible movement; stiffness is the prominent feature.

Deforming arthritis is more common in women than in men; like rheumatism, it may be excited by cold and damp, and is very apt to occur in the weak and unhealthy. It generally, even in cases that recover, persists for months. Nor will it yield to the remedies usually administered in acute rheumatism; nor to colchicum and the alkalies, so beneficial in gout. Its causation is still unsettled. In children a form of arthritis deformans has been particularly described by Still, in which with the general enlargement of the joints there is swelling of the lymph-glands and of the spleen.

I shall here add a short description of a disease of nutrition of dissimilar character to those described, but having this in common, that it markedly affects the organs of locomotion,—rickets.

Rickets.—In this country rickets is a comparatively rare affection, certainly rare as compared with its prevalence in England, in Holland, in Germany, and in some other Continental States. It is a constitutional disease of early childhood connected with impaired nutrition, and is chiefly characterized by increased growth of the epiphyses and periosteum, and imperfect ossification, producing softening of the bones with curvatures and distortions. The changes are most manifest in the long bones; and the amount of organic matter in them is more than doubled, while the calcareous salts are greatly diminished. Besides the osseous changes there is evident cachexia; and the liver and spleen become enlarged and indurated from overgrowth of the glandular elements and interstitial development of fibroid tissue. A similar process may also happen in the kidneys and in lymphatic glands.

Insufficient and improper food is a powerful cause of rickets. The malady may show itself as late as the seventh or eighth year; but it generally sets in during the first or second year of life. When it leads to death, it does so usually by gradual exhaustion, by impairment of the digestive functions, by thoracic complications, such as extensive bronchitis, pleurisy, or collapse of the lungs, by spasm of the glottis, by convulsions, or by chronic hydrocephalus. As a marked disease it does not usually last longer than a year, though the results of the osseous changes may long persist, and, affecting the thorax or the pelvis, prove eventually very injurious.

The beginning of the disease is generally about the period of dentition, and insidious. The child makes no attempt at walking, or ceases to walk if it have commenced. It is languid, irritable, its face pale, its tissues flabby. The appetite fails, there are thirst and irregularity of the bowels, or the marked signs of a gastro-intestinal catarrh. Restlessness at night, a disposition to throw off the bedclothes, pro-

fuse perspiration about the head, neck, and chest, while the rest of the body is hot and dry, attend an irregular febrile condition which soon shows itself; while fear of being touched, or general soreness and tenderness of the body or actual pain, bespeaks the local process that is going on in the bones and their covering. The changes in the bones now become more and more distinct. The joints appear swollen, especially at first the wrist-joints, and, when these are examined, the lower extremities of the radius and the ulna are found to be enlarged; similar changes are perceived in the tibia and fibula, and in the elbow. There is tenderness along the ribs, and, should the affection continue, nodules are felt at the junction of the ribs with their cartilages; the sternum protrudes, a pigeon-breast results; then the limbs show contortions, the clavicles are bent, the spine may be curved, the pelvis deformed. The head is large and square, the forehead high, the anterior fontanel remains unclosed, the sutures are open and thickened on the sides. A blowing sound is frequently to be perceived over the cranial sutures. Dentition is delayed, or the teeth decay and fall out. The urine is copious, and contains lactic acid and an excess of phosphates. Convulsions, laryngismus stridulus, and tetany are among the complications. In advanced cases the symptoms of cachexia are very marked; the flabby muscles, the wan, anæmic aspect, the large abdomen contrasting with the small face, the enlarged liver and spleen, the persistent tenderness over the bones, and at times the marked fever, give sad evidence of altered nutrition and of suffering; yet even then the little patient may recover, though most likely with part of the osseous system irretrievably damaged. Of course we have all kinds of gradations in the malady, and the general symptoms attending the morbid process may be slight, just as the rickety condition of the bones may be limited.

The *diagnosis* will have been made apparent from the description of the symptoms. In advanced cases there can be no doubt. The changes in the bones, the curvature, the distortions, the appearance of the patient, the evidences of cachexia, clearly stamp the malady. Earlier in the disease it may be confounded with the manifestations of *hereditary syphilis*. But this affection comes on even sooner than rickets, almost from birth; there are other signs of the constitutional taint, including early enlargement of the spleen, syphilitic coryza, and, at a later period, the notched teeth; a distinctive history may perhaps be obtained; and the enlarged bones not infrequently suppurate, the swollen epiphyses become detached, and osteophytes form,—changes not met with in rickets.

Mollities ossium produces deformities which may be mistaken for

those of rickets. But the softening of the bone is the result of its disease, and not of its want of proper ossification. There is considerable difficulty in locomotion, and the bones bend or break, after having been affected with deep-seated pains. The malady lasts for years, and is not one of childhood, being most common between the ages of twenty-five and forty, and attacking chiefly women. The pelvic bones are often implicated; it is doubtful if the phosphates in the urine are increased, but, as in rickets, the urine contains lactic acid. Yet there are not the characteristic signs at the cranial bones, the open fontanel and sutures, nor the swelling of the epiphyses, which this malady so strikingly presents.

There are cases described as acute rickets which are a combination of *rickets* and *scurvy*.[1] They are most common in infancy, and generally present the spongy gums only about the teeth that have been cut. They sometimes show, in addition to periosteal hemorrhages, a sudden protrusion of one eyeball. In the early stages rickets may be mistaken for *acute* or *subacute rheumatism;* the fever, the pain, the sweats, and the swelling near the joints mislead. But the age, the size of the epiphyses, the absence of redness of the joints and of heart-lesion, the "beading" of the ribs, the signs of beginning cachexia, the faulty dentition, and the pale urine full of phosphates, tell the true meaning of the symptoms. Moreover, the apparent joint-affection is apt to show itself at the wrist-joints, always a suspicious circumstance in delicate young children.

Some of the local deformities that result and the diseases with which they may be confounded, as of the thorax and of the head, have been elsewhere discussed. Besides the alteration of the skull in chronic hydrocephalus, the condition described by Elsaesser and others as *craniotabes* may be mistaken for ordinary rickets. It consists in thinning of the bones of the cranium, especially of the occipital bone, which becomes perforated, allowing the membranes of the brain to come in contact with the under surface of the scalp, and convulsions may be induced by undue pressure over the points of perforation of the bone. The malady, though regarded by some as a separate affection, is by others, by Virchow among them, looked upon as due to a rhachitic diathesis; we certainly often find evidences of this in conjunction with the peculiar alteration of the bones of the skull.

[1] Barlow, British Medical Journal, 1883, i. p. 1029, and "Bradshaw Lecture," *ibid.*, 1894; also St. Louis Courier of Medicine, 1883, p. 453.

CHAPTER XII.

FEVERS.

Fever is either a symptom of some strictly local malady or constitutes the only obvious affection present. In the latter case the disorder is called essential or idiopathic fever. The first step, therefore, when fever has been recognized, is to ascertain whether it is symptomatic or idiopathic; whether, in other words, it is but a complement to a disease, or, as far as can be ascertained, the disease itself. This is not generally a difficult matter. The history of the case, the course it takes, the absence or presence of the marked peculiarities of serious local disturbances, soon determine whether we are dealing with fever as a symptom, or fever as a disease. Idiopathic fevers, with some striking exceptions, are characterized by the want of definite and invariable anatomical lesions. That in all changes occur in parts of the nervous system, or in the blood, is highly probable. But there is no constant injury perceptible in the organs of the body: sometimes one, sometimes another, suffers; sometimes nearly all; at times, none, certainly none in an obvious manner. When we contrast this with symptomatic fever, the difference is striking. The visceral lesions, then, of an idiopathic fever are not the starting-point of the fever, but rather secondary and uncertain complications. In idiopathic fever, the fever controls the lesions; in symptomatic fever, the lesions control the fever. Idiopathic fevers are mostly infective and of bacillary origin.

Most fevers run a definite course, showing a strong tendency to a spontaneous termination at a given time. At their beginning, too, they are for the most part similar. There is a prodromic state, marked generally by unsound sleep, pain in the back, and lassitude. This is followed by chills, which are succeeded by heightened temperature, arrested secretions, quick pulse, and evident fatigue upon the least exertion. The fever now reaches its full development and its precise character becomes evident. After a while the disturbance declines, or speedily ceases under the influence of critical discharges, and a convalescence, more or less rapid, sets in. An unfavorable termination, on the other hand, may take place at any period after the system has been fairly invaded.

The marked features impressed upon the fever either by the course it runs, or by the specific nature of the symptoms, go to form what is called its *type*, and may be made the basis of the classification of all febrile disorders. But as opinions have been and are still diversified as to what really constitute the most palpable characteristics, so the classification of fevers is as yet, to a great extent, a matter of speculation. In the following table no attempt is made at an exhaustive or strictly scientific classification. Some disorders, such as cholera and epidemic dysentery, considered by many eminent pathologists to belong to idiopathic fevers, have no place assigned to them: pneumonia, notwithstanding its undoubted claims, has been already, for clinical reasons, elsewhere considered. Yet from a diagnostic point of view the arrangement adopted is convenient, and is sufficiently accurate to be free from grave objections.

Fevers.	
Continued Fevers	Simple continued fever.
	Catarrhal fever, or influenza.
	Typhoid fever.
	Typhus fever.
	The plague.
	Cerebro-spinal fever.
	Relapsing fever.
	Yellow fever.
	Dengue.
	Malta fever.
	Glandular fever.
Periodical Fevers, or Malarial	Intermittent fever.
	Remittent fever.
	Pernicious fever.
Eruptive Fevers	Scarlet fever.
	Measles.
	Rubella.
	Smallpox.
	Varicella.
	Miliaria.
	Erysipelas.

Continued Fevers.

All continued fevers are characterised by a steady progress of the febrile movement, without either decided exacerbation or remission, the rise and fall observable being too slight to modify the impression of a sustained action.

Simple Continued Fever.—Simple fever, or febricula, sets in with feelings of lassitude and dullness; in these subsequent but scar-

excited pulse, thirst, headache, pain in the limbs. The bowels are generally confined, the urine high-colored. The fever is soon at its height; it then either gradually declines, or is more suddenly relieved by copious perspiration or by a critical discharge from the bowels. Generally it runs through all these stages in a few days; but it may be protracted for upward of a week or longer. On the other hand, a day may witness both its beginning and its termination. The convalescence is almost always rapid.

The exciting causes of this form of fever are fatigue, errors in diet, change in mode of life, exposure to cold and damp, or to the sun, and there is no doubt that ptomaines may also act an important part in its production. When brought on by mental overwork or by anxiety or grief, it is not uncommonly attended with increased sensibility of the skin, and with considerable prostration, simulating typhoid fever, but differing from it by the absence of epistaxis, of the peculiar abdominal symptoms, and of the eruption. More frequently the fever has the appearance of one of high action. At times, indeed, it is so intense, and the vascular system is so wrought up, that the distemper assumes what is called an inflammatory type. It then exhibits the characteristics of the fever described by the physicians of the last century as synochus. A temperature of 103° or upward, throbbing of the temporal arteries, severe headache, and delirium are among its symptoms. This variety of the fever is not now encountered, save in tropical latitudes, and is a form of the so-called thermic fever of Guitéras. In point of diagnosis, it is most apt to be confounded with internal inflammations, especially with meningitis. But there is not the vomiting, nor the irregular pulse this presents.

In addition to these ordinary forms of simple continued fever, which are of short duration, there is a form, rare it is true, of very long duration, and in which the fever may last for weeks, without internal complication or obvious cause. The absence of eruption, of enteric symptoms, and the negative character of the Widal test distinguish them from typhoid fever.[1]

Catarrhal Fever.—This epidemic malady, which belongs to the idiopathic fevers, is sometimes described as a mere variety of bronchitis, because inflammation of the bronchial mucous membrane constitutes one of its most prominent symptoms. But this is not a just view. With as much reason might typhoid fever be omitted from the list of febrile maladies and described as a variety of enteritis.

[1] See a paper of mine with illustrative cases in Amer. Journ. Med. Sci., June, 1896; also Heubner, Deutsches Archiv für klinische Medicin, vol. lxiv., 1899.

Catarrhal fever, or influenza, is essentially an epidemic disease, the history of which is not confined to any particular time or to any particular nation. Its cause is believed to be a slender bacillus found in the expectoration and nasal secretion.[1] But its bacillary origin, though very probable, has not been demonstrated beyond question. Each epidemic does not furnish precisely the same train of symptoms; but they all agree in this: the disorder sets in suddenly and attacks pre-eminently the mucous membranes. Generally it is the mucous membrane of the nose, eyes, and bronchial tubes that suffers most, and we find the signs of coryza and of bronchial inflammation,—a watery eye, sneezing, uneasiness about the throat, and a tormenting cough. But associated with these are great depression of spirits and usually an extraordinary amount of lassitude and impairment of strength,—much more than the cold in the head, or the laryngitis, or the bronchitis, will account for. The skin is hot, at times covered with perspiration; the thermometric record is peculiar only in its extreme irregularity. The temperature generally ranges between 100° and 102°, or starts up suddenly to 104° or 105°, and in less than a day subsides almost to normal; the pulse is of moderate volume, the tongue coated; the patient complains of debility, of headache, of aching pains in his back and limbs, and of constriction at the lower part of the chest. Often there is some dyspnœa, as well as epistaxis, hyperæsthesia, especially of the neck and head, and disturbance of the alimentary tract, evinced by loss of appetite, nausea, and vomiting, or by diarrhœa. Commonly after three or four days these symptoms begin to subside, the cough and debility outlasting the other morbid signs. The cough is often dry and harassing, and chiefly laryngeal.

But all epidemics do not run precisely this course. In some, the prostration is not so evident, and the febrile signs are more active and of an inflammatory type; in others, the pain and soreness in the limbs and in the joints are the most prominent symptoms; or we may find hemicrania, or torpor and delirium, or parotitis with salivation, or otitis, or epistaxis, or catarrhal jaundice, or bronchitis of the finer tubes, or pneumonia, or tendency to heart-failure, or meningitis, basilar or spinal, and irregular rashes, as complications. Further, as complications or sequelæ of influenza have been observed various psychoses and neuroses, or neuritis, local or multiple, bulbar palsy, acute ascending paralysis, hemiplegia, diabetes, vascular occlusion, gangrene, angina pectoris, inflammation of the lymphatic glands and

[1] Pfeiffer, Zeitschrift für Hygiene und Infektionskrankheiten, March 3, 1893.

of the antrum, acute nephritis, and painful and inflammatory affections of tendons, fasciæ, joints, periosteum, and bones. The disease also brings out a latent syphilitic taint.[1]

The lung complication of influenza is striking. It is mostly an intense congestion, with bronchitis, here and there with spots of consolidation, a broncho-pneumonia. True lobar pneumonia is much rarer. The lung affection may be of long duration, showing the record of a fever with marked rises and remissions. After declining, the temperature may become subnormal and remain so with occasional exacerbations for a long time, as seen in the accompanying chart of a case in my ward at the Pennsylvania Hospital.[2]

Influenza is not ordinarily in itself a fatal disease. It is only so in the very young or the very old. It is also a grave malady in persons with weak hearts. A source of danger is the indurated lung it may leave behind becoming the seat of tuberculosis.

Catarrhal fever is easily discriminated from other maladies. Its peculiar epidemic character and the prostration prevent us from mistaking it for an ordinary cold or bronchitis. Occasionally the attending debility makes it look like the onset of a long-continued fever. But brain-symptoms are present only in rare instances in influenza; and, on the other hand, decided catarrhal symptoms are not common in typhoid and typhus fevers. Before long, too, the eruption of these diseases clears up whatever doubt may have existed; rashes of any kind are extremely rare in influenza, and are of irregular type when they happen. At times there is a long-continued fever in influenza like that of typhoid fever, but the Widal reaction of this is lacking.

Catarrhal fever may be mistaken for *hay-fever*. But the local symptoms of irritation of the nostrils, the watery eyes, and the reddened conjunctivæ are very striking, and the febrile movement is generally less than in catarrhal fever. Moreover, there are asthmatic symptoms in hay-fever or hay-asthma in a certain proportion of cases; and the history of the case, the manner in which it comes on as a rose-cold in the latter part of May or early in June, or as autumnal catarrh after the middle of August; the hereditary idiosyncrasy so often seen; the persistence of the attack while exposed to the peculiar vegetable emanations that give rise to it; its almost abrupt cessation on removal to certain localities,—make up a set of features which are very distinctive.

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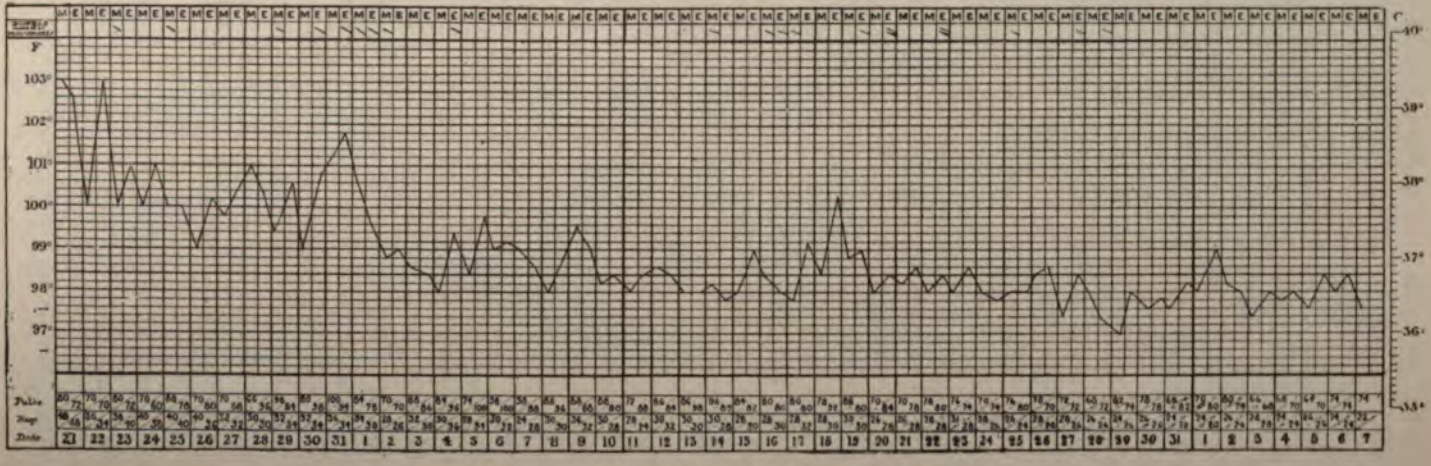
[1] Howard, Lancet, July, 1899.

[2] For a full description of the lung complications of influenza, see my paper on the subject in the "International Clinics," Vol. I., Second Series.

FIG. 74.



Temperature in a case of influenza with intense congestion of the lungs.

When influenza is prevailing on a large scale, it is often found masked by other diseases, and it may be difficult then to separate its manifestations from those of the malady it accompanies. Other peculiarities of influenza are the long time it takes the patient to regain his strength, and the annoying sweats that attend convalescence. This was striking in the epidemic of the early winter months of 1890; as was also the tendency to repeated attacks, to irregular heart action, and to alterations of cutaneous sensibility.

Typhoid Fever.—In this country and on the continent of Europe a form of continued fever prevails, especially among young adults, that is marked by great prostration and disturbance of the nervous system, and by constant anatomical lesions. To this disease the designations of typhoid fever, enteric fever, and abdominal typhus have been applied.

The distemper may set in suddenly, but more generally it has an insidious beginning. For some days preceding the access of the fever the patient feels weak. He is without animation, complains of soreness and fatigue, of dull pain in the head, of loss of appetite. His sleep is unsound; all exertion is wearisome. A fever now appears, preceded mostly by a chill, or by chilly sensations, which alternate with flushes of heat. The muscular prostration accompanying the febrile movement becomes so great that the patient is obliged to keep his bed. His appetite is entirely gone, the tongue is coated, the bowels are loose, the abdomen is somewhat swollen and tender to the touch.

The malady has now completed its first week. It enters on the second week with fever unabated, and with the signs of disturbance of the alimentary tract and of the nervous system more and more unmistakable. There is sometimes nausea or epigastric distress, often pain in the right iliac fossa, increased by pressure and tympanites. On close inspection, a few reddish spots, resembling flea-bites, are found on its surface. The tongue dries and becomes reddish or brownish; it is often glazed and covered with a light coat; sometimes it has deep fissures; very frequently I have noticed at the tip a wedge of brownish or reddish surface free from coat, but which begins to be covered over as the disease declines; the gums and teeth are lined with dark crusts. The mind is dull and wandering; cough and great restlessness exist; the debility is extreme.

The disease now begins to draw to its close. It has reached the third week, and a change, for better or for worse, may be looked for. Slowly recovery sets in, marked by a brightening of the countenance and by a gradual increase in consciousness and strength; or deepen-

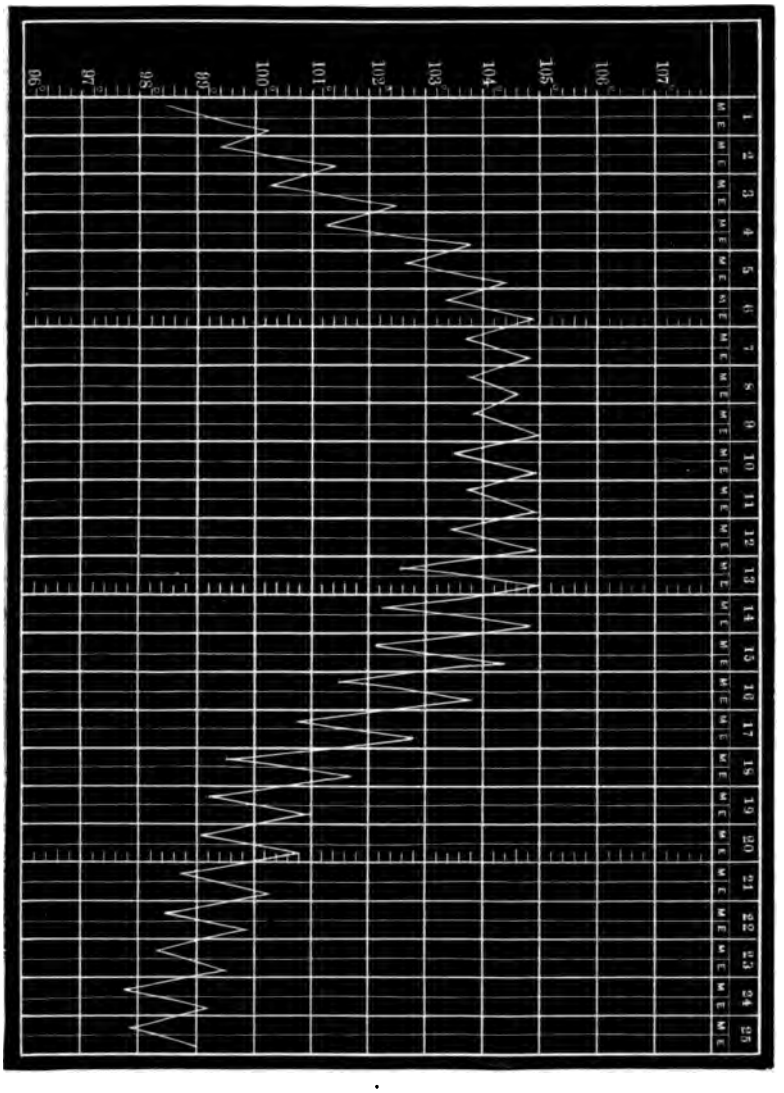
ing insensibility, jerking of the tendons, feeble pulse, and cold, clammy sweats indicate that dissolution is fast approaching.

Thus, in one way or the other, the fever itself is apt to terminate by the beginning or, more generally, by the middle of the fourth week. Yet such is not always the case. Death may take place at an earlier period; or, on the other hand, the malady, by troublesome complications, may be lengthened beyond the second month. Under any circumstances, convalescence is protracted. The nervous system rallies but gradually from the shock it has received.

Among the symptoms enumerated, some tend clearly to characterize the disease. And, first, of the more purely febrile symptoms. The *skin* during the fever is mostly dry. But there may be an acid perspiration, very manifest during the whole course of the disease, and also encountered long after convalescence has set in. The *pulse* is accelerated, mostly about 120, and is rapid even after the fever has left, though in convalescence it may be much slower than normal; it is very compressible, and, intercurrent acute inflammations notwithstanding, it seldom loses its compressibility. A jerking, irregular beat, or very great rapidity, a running pulse, is an unfavorable sign. Dicrotism of the pulse is not unusual. Associated with the diminished strength of the pulse is a decided faintness of the first sound of the heart.

The *temperature* is peculiar; in the first five or six days of the disease it pursues an ascending line; that is to say, starting at the normal 98.6° F., there is a daily evening rise of about 2°, with a morning remission of about 1°. From the fifth or the sixth day to the twelfth or a little later,—roughly speaking, we may say from the end of the first week to the end of the second,—the fever is continuous, with a morning remission rarely exceeding 1°. From that time on, let us say from the twelfth day, although the evening temperature may remain for a day or two quite or nearly as high, there is an abatement of from 1° to 2° in the morning. These changes between morning and evening become very evident at the end of the week, and are still more evident in the third week, when the morning and evening temperatures may vary as much as from 4° to 6°. During this week, too, the evening temperature gradually decreases; but in severe cases it remains high, and there are no decided remissions either in the second or the third week. The morning temperature is high, 104° or more, and there may be still greater heat of skin in the evening, or else it differs but little from that of the morning. One hundred and six degrees is a high temperature, but I have known it 107.5°, yet the patient recover. The peripheral tem-

perature, as measured, for instance, in the palm of the hand, becomes during the fever as high as the axillary temperature, but their equali-



Temperature chart in a case of typhoid fever of decided kind.

FIG. 75.

zation ceases prior to defervescence.[1] In exceptional instances, the temperature may be normal throughout;[2] in still rarer instances it

[1] Couty, Archives de Physiologie, No. 2, 1880.

[2] Vallin, Arch. Gén. de Méd., Nov. 1873; Finlayson, American Journal of the Medical Sciences, March, 1891, p. 225; Wendland, Deutsche Medicinische Wochenschrift, Aug. 29, 1892; Dreschfeld, Practitioner, No. 298, vol. l., p. 272; Fisk,

is subnormal.[1] I have never seen a case of either kind. Occasionally the curve may resemble that of intermittent fever.[2] Again, the fever may terminate by crisis at the end of the third or in the fourth week. This I have met with more than once.

The *urine* is acid, high-colored, scanty,—the urine of fever. Ehrlich has stated that the urine of typhoid fever gives a special reaction,—the diazo reaction. This test consists in taking forty parts of a saturated solution of sulphanilic acid in hydrochloric acid, one to twenty, and one part of a one-half per cent. solution of sodium nitrite, and adding them to an equal bulk of urine rendered alkaline by strong ammonia. Normal urine is colored brownish by the test liquid, typhoid-fever urine pink or ruby, with slight frothing. The reaction has not been found in all cases of typhoid fever, and has been obtained in a variety of other morbid conditions, such as tuberculosis, typhus fever, measles, scarlatina, enteritis, malaria, pneumonia, meningitis, septicæmia, uræmia. The toxicity of the urine is greatly increased in typhoid fever. Robin[3] regards the urine as characteristic even from the onset; the chief characteristics being a peculiar odor, constant presence of albumin in moderate amount, absence of urohæmatin, presence of indican, increase of uric acid, marked diminution of the earthy phosphates. As regards the albumin, I do not think it constant, and it is not in large amounts. It is most marked in severe cases and those with high temperature, and coexists with a few tube-casts. In the so-called *renal* type of typhoid, in which an acute nephritis for the most part happens, we also find red corpuscles, free granular epithelium, and casts of various kinds, though not oily; there is considerable albumin, and often at the onset a scanty bloody urine. The kidney involvement may manifest itself from the start, and persist throughout. In very rare instances of typhoid fever there is marked hæmaturia.

Among the abdominal symptoms, *diarrhœa* is prominent. It is mostly present, except when the disease is unusually mild, though its prevalence varies in different epidemics. Generally it is a very early symptom; at times it is even seen among the prodromes. The clue to its cause is found in the state of the intestinal glands, in the enlargement and ulceration of the glands of Peyer and of the solitary

Medical News, Nov. 3, 1894, p. 479; MacDougall, Lancet, April 15, 1893; Memphis Lancet, July, 1898.

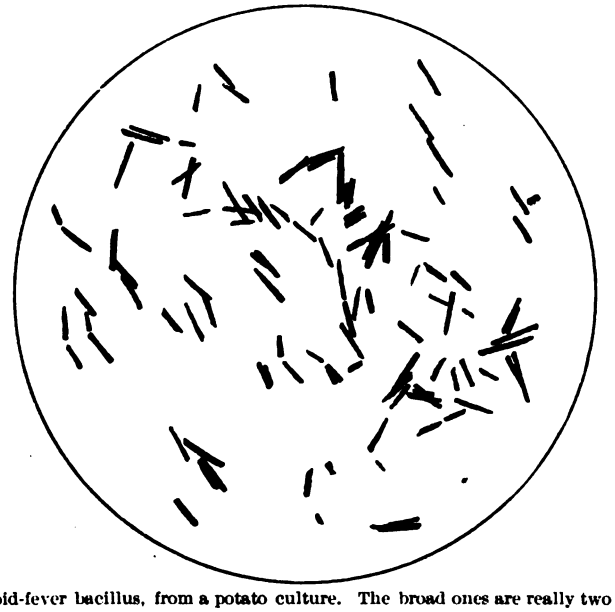
[1] Raimondi, Gazette des Hôpitaux, 1894, No. 109; Centralblatt für Innere Medicin, 1895, No. 6, p. 152.

[2] MacDougall, Lancet, April 15, 1893.

[3] Bulletin Méd., No. 87, 1897.

glands, with the tumefaction of the mesenteric glands. And in these morbid alterations we find an explanation not only of the occurrence of the diarrhœa, but also of its frequency. The stools are thin, of a yellow or dark-brown color, and of offensive smell. When the affection is at its height, from three to four evacuations occur during the twenty-four hours; but the passages may become much more numerous, and with their number the danger rises. If they take place without the knowledge of the patient, his situation is precarious. Sometimes the stools contain blood. Should this be present

FIG. 76.



Eberth typhoid-fever bacillus, from a potato culture. The broad ones are really two bacilli lying in juxtaposition. *Zeiss* $\frac{1}{12}$, *homo. im.*, Oc. 5.

in considerable quantity, it is a very unfavorable circumstance. Yet intestinal hemorrhage is by no means necessarily fatal. In rare instances there is hæmatemesis, in others hæmoptysis, and this in the absence of any pulmonary lesion.[1]

The bacillus giving rise to typhoid fever is the bacillus typhosus described by Eberth and by Gaffky (Fig. 76). The bacillus is chiefly found in the intestinal lesions and in the stools. But it is very generally diffused, being met with in the gall-bladder, in the bone-marrow, in the blood, in the coats of vessels, and in the urine even in cases in which there are no renal symptoms. It is a rather plump

[1] Dickinson, Lancet, Feb. 17, 1894, p. 421.

organism from two to three μ long, with rounded ends, actively motile, and staining with the ordinary aniline colors. It is very sensitive to high temperatures, and does not form spores.

Unlike the colon bacillus, the typhoid bacillus does not cause fermentation in glucose-containing solutions, nor does it curdle sterilized milk; it grows readily on gelatin plates. Potato cultures of the typhoid bacillus are, as a rule, scarcely visible, while those of the colon bacillus appear as distinct, broad, orange streaks. Finally, the typhoid bacillus does not yield the indol reaction with the ordinary tests, while the colon bacillus does.

For diagnostic purposes the most valuable property connected with the bacillus typhosus is the arrest of motility and the agglutination occasioned when brought in contact with immune serum or a culture of the typhoid bacillus,—the Widal test. The clumping that occurs is characteristic, and happens in from one to fifteen minutes with a dilution of 1 to 10. The applicability of the test has been immensely widened by the use of dry blood, as suggested by Wyatt Johnston; it is thus largely employed for purposes of public sanitation. A drop or two of blood is collected on glazed paper and can be transmitted to a laboratory by mail, and is for a long period ready for testing by simply dissolving the dried blood in water.[1]

The Widal reaction is the most important of all recent additions to the diagnosis of typhoid, or, indeed, of any idiopathic fever. I have used it very extensively at the Pennsylvania Hospital, and believe in its wide applicability. It gives accurate results in fully ninety-two per cent. of the trials, if the technique be careful, and many of its supposed failures are due to defective technique. It has, however, its limitations. If a person has once had typhoid fever the reaction may show itself for years afterwards, and be very misleading should a fever attack of doubtful character occur. Then it is of little value in the first week of the disease, rarely being manifest before the fourth day, and often not until the sixth to the eighth day. It may not show itself until very late in the disease. Thus in one of my cases of renal typhoid, repeatedly tested, there was no Widal reaction until in the fifth week. It is sometimes obtained where there is no clinical evidence of typhoid fever, though we must remember, especially in hospital practice, the possibility of the patient having had this years before. I have found most of these erroneous tests to be in instances of acute rheumatism, of acute tuberculosis, and of nephritis.

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[1] For the exact and fuller technique, see works on bacteriology, or "The Diagnosis of Disease," by Cabot, 1899.

Enlargement of the spleen is a very constant attendant upon the fever. The tympany that often exists interferes with the recognition of the enlargement.

Another abnormal symptom of significance is *pain*. It varies much in severity and character, and is, indeed, not always present. It is often a heavy, aching feeling. In some patients it is of a griping kind, preceding the loose discharges; in others it seems to be called into existence only by pressure. Its most common seat is in the iliac fossæ, especially in the right iliac fossa, and the pain corresponds, for the most part, to the seat of the lesions. In rare instances the pain is really in the muscles, which may, indeed, suppurate.[1] Often, while the hand is exploring the abdominal regions, a movement of the fluid and gas in the distended bowel, attended with a gurgling noise, becomes appreciable. This sign is best elicited near the ileo-cæcal valve.

During convalescence, griping pains are not infrequently complained of. They are colicky pains, produced generally by errors in diet, and may be followed by a return of the diarrhœa. But pains at almost any stage of the disease may be also due to peritonitis and to perforation.

Hardly inferior to the abdominal symptoms in import are the signs of *disturbance of the nervous system*. The fever is, as its old name implies, pre-eminently a "nervous" fever: the nervous symptoms are, in truth, never absent; but, though always present, they are less extensive in some cases than in others, and not the same throughout all the stages of the disease. Thus, early in the disorder, dull headache, mental languor, wakefulness, and a perverted state of the senses, such as ringing in the ears and dulness of hearing, are encountered; while later, great restlessness, delirium, somnolence, or coma, and jerking of the tendons are phenomena more likely to be met with. Occasionally the disease is ushered in by acute mania.[2] In some epidemics the nervous symptoms are so pronounced that a cerebro-spinal type of the disorder is recognizable.

The *delirium* sets in generally during the second week, for the most part at night, and terminates with convalescence or ends in coma. It is not a wild delirium, but a confusion of mind associated with rambling thoughts. If the patient's attention be strongly engaged, he may be almost always roused, and does for a time as he is

[1] Ebing, Archiv für klin. Med., viii.

[2] Hare and Patek, Medical News, June 20, 1891, p. 681; MacDougall, Lancet, April 15, 22, 1893.

told; but after a short interval his muttering lips indicate that some curious fancy has again taken possession of him. In some cases, not in many, the delirium is attended with great restlessness and agitation, and the sick man, if not prevented, attempts to walk about the room. This kind of frenzy often ends in fatal coma. Equally unpromising is early or unremitting delirium. When contrasted with the mental wandering in other acute disorders, the delirium of typhoid fever exhibits peculiar traits. It is ordinarily more active than that of typhus; far less demonstrative or talkative than the mania of drunkenness; as aimless as, but less continued than, the ravings of inflammation of the brain. Great restlessness and tremors, associated with a clear mind, and at times with copious perspirations, have a very significant meaning: they point to deep and extending ulceration.

Other symptoms of grave disturbance of the nervous system show themselves in violent general *convulsions*. These are more common in children than in adults, in whom they may be a late symptom; they may or may not be of uræmic origin. The knee-jerks are present, unless peripheral neuritis exists. In severe cases both the reflexes and the muscular irritability are said to be increased.[1] On the other hand, it has been observed that in children the tendon-reflexes are often enfeebled during the acute stage of the disease and exaggerated during convalescence.[2]

In some cases of typhoid fever symptoms not only cerebral but also of spinal origin appear, and they may, indeed, assume a high degree of intensity. We find extensive cutaneous hyperæsthesia, spinal pain and tenderness, with a sense of pricking along the vertebral column, and, in some instances, cutaneous and muscular anæsthesia, numbness of the extremities, partial paralysis or convulsive contractions of the respiratory muscles, convulsive cough, paralysis of the sphincters, contractions of the extremities, and even rigidity of the muscles of the neck. These spinal symptoms are more common when the disease is epidemic than when it is sporadic, and are always indicative of a very serious form of the disorder. They sometimes persist after the fever has left, or indeed—and this is especially true of paralysis—may not appear until convalescence. The *palsy*, the most common form of which is paraplegia, mostly begins gradually and disappears gradually. It may be preceded by trembling movements, suggesting disseminated sclerosis; but the tremor is rather the result of general debility, and is not associated

[1] Angel Money, Lancet, Nov. 7, 1885, p. 842.

[2] Albouze, Journal de Médecine et de Chirurgie Pratiques, Sept. 10, 1892.

with difficulty of enunciation. There is much evidence that the paralysis after typhoid fever is due to multiple neuritis.[1]

Hawkins[2] has reported a fatal case of typhoid fever complicated by intestinal hemorrhage and purpuric spots and the development of right hemiplegia and aphasia due to occlusion of the left middle cerebral artery. He has collected seventeen cases of typhoid fever complicated by hemiplegia. Most often the palsy was right-sided and associated with aphasia. Usually there was recovery from the paralysis, but in three cases this was persistent. The complication was most common at the end of the attack or during convalescence.

Two other prominent symptoms of the malady must still be inquired into: one is epistaxis; the other, the cutaneous eruption. *Epistaxis* is not often absent in grave cases. It may happen at any period; but it generally takes place before the disorder is far advanced. The quantity of blood lost is rarely considerable; and for this reason the bleeding is frequently overlooked.

The *eruption* peculiar to the disease is the rose-colored rash. It appears on or shortly after the seventh day, but occasionally not until the end of the second week. It consists of small, red spots, only very slightly elevated above the skin, somewhat similar to flea-bites, yet differing from them in lacking the central mark and in their finer, paler color and less obvious outline. The spots are seen upon the abdomen and chest, rarely upon the extremities, almost never upon the face. They disappear totally on strong pressure, yet return immediately when the pressure ceases; their disappearance and reappearance are best studied by stretching the skin. They are generally few in number, and not persistent. Each spot does not last for more than three or four days; then it fades, and a fresh one near by replaces it, and runs the same course. Spots thus appear and pass away for more than a week, after which, in most cases, they entirely vanish. During convalescence not a trace of them can be found. The eruption, although very common, is not invariably present; at all events, it is not invariably found. Beyond doubt, too, it is in some epidemics more constant and marked than in others. Late in the disease another eruption appears, consisting of minute transparent vesicles, scattered plentifully over the body. These sudamina are not so frequently encountered as the rose-rash, and are not characteristic. As further, though rare, eruptions in typhoid fever we find blue spots, a

[1] Pitres and Vaillard, Rev. de Méd., 1885, t. v.; Ross, Amer. Journ. Med. Sci., Jan. 1889; Bury, Medical Chronicle, June, 1892.

[2] Transactions of the Clinical Society of London, vol. xxvi., 1893, p. 50.

scarlatiniform, or a measly rash.[1] There may be also, as in typhus, mottling of the skin, and the cerebral spots are readily produced. Attention has been called to a yellowish discoloration of the palms of the hands and soles of the feet in cases of typhoid fever.[2]

The blood in typhoid fever shows constant reduction in hæmoglobin, and a relatively smaller diminution in the red corpuscles. But the most characteristic point connected with it is, as we know chiefly through the admirable researches of Thayer, that the white corpuscles remain normal or are slightly diminished. The absence of leucocytosis or the hypoleucocytosis becomes, indeed, a valuable sign, and distinguishes typhoid fever from acute inflammatory conditions and from septic fevers. Nay, it has been found that even where suppurating complications exist, such as otitis media, streptococcus pneumonia, decubital abscess, there is no increase of the leucocytes above the normal.[3] Osler has, however, observed an increase in the polynuclear forms where an acute inflammatory process occurs in typhoid fever, as in connection with perforation.

After convalescence has set in, we may have a return of fever. It may be either a transitory and slight return, due to fatigue or to some indiscretion in diet, or a more protracted state, in which most or all of the symptoms peculiar to the disease reappear. Thus, typhoid fever *relapses* usually come on in the second week of assured convalescence, and, according to my experience,[4] occur suddenly; soon diarrhœa, furred tongue, and enlargement of the spleen are manifest, and on the fourth or fifth day reappears the characteristic rose-rash, which is often somewhat coarser than in the first attack, and does not show the same disposition to appear in successive crops. With the eruption delirium often comes back. The temperature is unlike that of the original attack in quickly reaching a high point of fever-heat; after the first day or two it remains more or less stationary, with a slight morning fall, for five or seven days usually, and then shows the well-known remissions and rises of the zigzag decline. The relapse is in its duration usually much shorter than the original attack, and generally, notwithstanding the threatening appearance of the symptoms, ends in convalescence. During its progress intestinal hemorrhage may happen; and after return to apparent health a second relapse or more

[1] For a description of these anomalous rashes, see a paper of mine, Amer. Journ. Med. Sci., July, 1899.

[2] Filipovitch, Lancet, Aug. 19, 1893. See also Med. News, Oct. 4, 1893, p. 444.

[3] Kölner, Deutsch. Arch. f. klin. Med., Bd. lx.

[4] See article on Relapses of Typhoid Fever, Transactions of the College of Physicians of Philadelphia, 1877.

may occur. Each relapse occasions characteristic markings on the nails, from impaired nutrition, which Longstreth has very fully described.[1] Ziemssen specifies the fifth, seventh, and fourteenth days after the cessation of the original fever attack as the days on which a relapse is likely to happen.[2] The temperature sometimes keeps up a degree or two, while the patient is in every other respect fully convalescent, yet will come speedily to the norm if he be made to leave his bed.

Both during the height of the fever and in convalescence, but more especially during the latter, *certain complications or sequelæ* may arise, some of which are medical, such as parotitis, swelling of the submaxillary glands, otitis media, erysipelas, noma, laryngeal ulceration or stenosis, milk-leg, thrombosis of the femoral artery, the result of arteritis,[3] jaundice, acute cholecystitis, abscess of the liver, periostitis, osteomyelitis, gangrene of the skin,[4] transitory aphasia,[5] hemiplegia, paraplegia, hysteria, local neuritis, tenderness of the toes, pseudo-dementias, and insanity; while others, as dislocations, caries, necrosis of bones, epididymitis,[6] orchitis,[7] vesicular catarrh, abscess, and gangrene, come within the domain of surgery.[8]

Among the medical complications a few must be specially examined into, as they may involve grave questions of operative procedure. To these belong some of the *hepatic complications*. Now, while jaundice is a rare symptom in enteric fever, hepatic complications are not. I have collected fifty-five cases of jaundice, eight of which were my own,[9] and they occurred in various conditions,—some, not many, were catarrhal, others were connected with abscess or with acute yellow atrophy of the liver, or with pylephlebitis, or with acute cholecystitis; the majority were of blood origin. By far the largest number of hepatic disorders in typhoid fever occur without jaundice, and are

[1] Relapses of Typhoid Fever, Transact. Coll. of Phys. of Phila., 1877.

[2] Arch. f. klin. Med., Feb. 1884.

[3] Lucas-Championnière, Journ. de Méd. et de Chir. Pratiques, 1888.

[4] Stahl, Phila. Med. Journal, Oct. 1898, and Transactions of the College of Physicians of Philadelphia, 1899.

[5] Arch. f. klin. Med., Bd. xxxiv., 1, 1883.

[6] Girode, Archives Gén. de Médecine, Jan. 1892, p. 43.

[7] Eshner, Phila. Med. Journ., May, 1898.

[8] See an admirable discussion of these surgical complications in the work, "On the Surgical Complications and Sequelæ of Typhoid Fever," 1898, by Dr. W. W. Keen.

[9] See papers "On the Significance of Jaundice in Typhoid Fever, and on the Hepatic Complications without Jaundice," in Amer. Journ. Med. Sci., July, 1898, and "On Cases of Cholecystitis ending in Recovery," *ibid.*, Aug. 1899.

very difficult to recognise,—may be, indeed, entirely latent. This is especially true of the gall-bladder infection, and the secondarily induced cholecystitis. Gall-bladder infection is so common in typhoid fever as to be the rule: gall-bladder symptoms are the exception. Yet cholecystitis, if looked for, is oftener found than appears at first sight. Its symptoms are the same as when not due to bacillary typhoid infection: severe pain, tenderness, rumor, nausea, and vomiting.

The appendix is not infrequently the seat of typhoid ulcers, and they may even lead to perforation. Yet *appendicitis* with distinct symptoms, and as a recognizable complication, is a very rare disease. But I have seen several instances of it. The localized tenderness at or near McBurney's point, a peritonitis spreading from there, rigidity of the right rectus muscle, sense of resistance or circumscribed tension in the right iliac fossa, and vomiting are the most significant symptoms. Deaver[1] lays stress on the nausea and vomiting ceasing when the pain becomes localized in the right iliac fossa: there may or may not be the history of a previous attack. The symptoms may continue for a considerable time and slowly subside, or perforation, or an abscess follow. Hare[2] has recorded a case in which in the course of typhoid fever a perityphlitic abscess formed, and in which recovery followed an operation. There was decided leucocytosis.

The worst of the complications of typhoid fever is *perforation*. This occurs in from two to two and a half per cent. of the cases, and it is asserted to be rather more common when the bath treatment has been employed. It is much more usual in men than in women, and is very generally fatal. The accident usually happens at the end of the third week, or later, though it is not extremely infrequent in the second week. At times the symptoms of perforation are latent, and masked by the general gravity of the case, and the great meteorism: it is only at the autopsy that the perforation is found. When perforation occasions symptoms, these are of two kinds: there is either collapse followed by peritonitis, or there is a spreading peritonitis without the signs of collapse. In the first case we meet with sudden acute abdominal pain, referred to the lower part of the abdomen, vomiting, signs of prostration, pinched features, rigidity of the recti muscles, at times fall of temperature, and subsequently chills, elevated temperature, local tenderness, followed by spreading peritonitis. In the second case the peritonitis alone is marked, and is attended or not with tympanitic distention or vomiting or sweats, but always with

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[1] Amer. Journ. Med. Sci., Jan. 1895.

[2] Medical Complications and Sequelæ of Typhoid Fever, 1894.

obviously increasing gravity of the case. Under either set of circumstances great diminution of the hepatic dulness is a valuable sign. To this may be added marked leucocytosis, as found by Thayer. The same symptoms as those of intestinal perforation may be due to perforation of the appendix or of the gall-bladder in typhoid fever, and no distinction is possible, unless the exact seat of the pain and of the early peritonitis and the history of the case enable us to make it.

Occasionally, unfortunately not often, inflammatory adhesions close the perforation and recovery ensues without an operation; or a localized abscess results. The peritonitis and intestinal adhesions that follow perforation may be attended with symptoms of obstruction of the bowel.[1]

Sometimes sequelæ appear long after the primary disease has come to an end. Orlow[2] has reported a case in which five and one-half months afterwards typhoid bacilli were detected in a granuloma of the tibia. Péan and Cornil[3] observed a case in which five months after a typhoid-fever attack typhoid bacilli were found in the lesions of a suppurative periostitis, and Van Dungern[4] reports an instance of typhoid bacilli met with in the pus from an abscess around the gall-bladder fourteen and a half years after the attack. Sudden death may take place in the course of typhoid fever as a result of disturbances in the circulation, from the formation of blood-clots, from inflammatory and degenerative changes in the muscular wall or disorder of the nervous supply of the heart, or from the poisoning of the system that is an essential part of the disease.[5] Death has also resulted from profuse sweating.[6]

The disorders with which typhoid fever may be confounded are not the same at all the stages of the complaint. Early in the affection it is most likely to be mistaken for simple continued fever, for influenza, or for one of the exanthemata. But diarrhœa is not present in these, nor are there marked prodromata; and whatever doubt may exist with reference to the first two is cleared up in a few days, since the temperature-record is different and the acute symptoms generally come to an end at a time at which in typhoid fever they begin to be more and more developed. The exanthematous fevers

[1] Blaikie Smith, International Clinics, vol. i., 2d Series, 1892, p. 79.

[2] Deutsche Medicin. Wochenschrift, Nov. 27, 1890.

[3] Bull. de l'Académie de Médecine de Paris, April 14, 1891.

[4] Münch. Med. Wochenschr., 1897, No. 26.

[5] Dewèvre, Archives Générales de Médecine, Oct., Dec. 1887; Galliard, *ibid.*, May, June, 1891.

[6] Juhel-Renoy, Archives Générales de Médecine, 1886, vol. i. p. 274.

cannot, before their eruptions appear, be distinguished with absolute certainty; though we may suspect measles by the coryza, scarlatina by the sore throat, and smallpox by the lumbar pains and high fever.

At a more advanced period, typhoid fever may be confounded with typhus, and with these morbid states:

General Debility:

Typhoid Conditions:

Enteritis:

Peritonitis:

Appendicitis:

Meningitis:

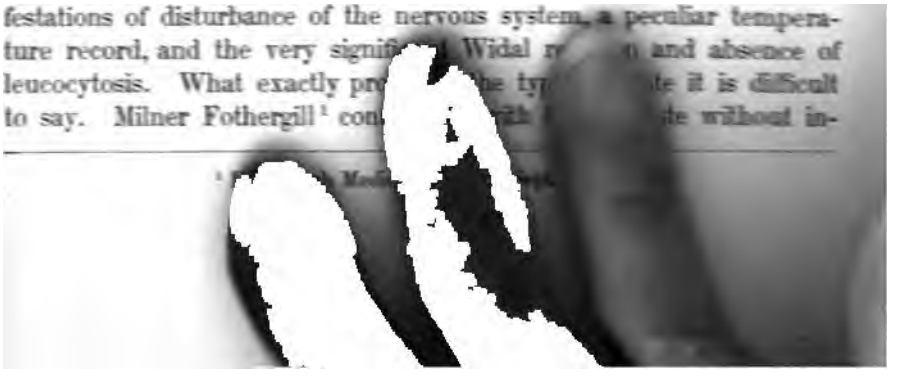
Ulcerative Endocarditis:

Acute Pulmonary Affections.

General Debility.—It does not seem likely that so acute and dangerous a malady as typhoid fever could be mistaken for mere debility: yet such an error may occur when the disease is latent, or so light as not to confine the patient to bed. In these so-called "walking cases" the debility, however, sets in suddenly, and not gradually, as in weakness from general constitutional causes. Moreover, the abdominal symptoms are rarely wanting, and there is more or less confusion of mind. The thermometer is of signal value. But the greatest safeguard against error is to be aware that the disease assumes at times a latent form, and to examine every case of sudden debility, to see if under its mask are hidden the features of typhoid fever.

Typhoid Conditions.—No blunder is more common than to misconstrue into typhoid fever a typhoid condition of the system. We may find this condition in many different complaints, both acute and chronic; but more especially are purulent infection, some forms of pneumonia, dysentery, erysipelas, and abscess of the kidney attended with delirium, drowsiness, dry, brown tongue, and extreme prostration,—in one word, with a typhoid state.

Yet a typhoid state is not typhoid fever: it is simply a low condition of the system which may be present in many dissimilar maladies, and which is present in its most perfect form in typhoid fever. But in this disease we have other signs than those of adynamia: we find joined to it diarrhœa, tympanites, epistaxis, an eruption, special manifestations of disturbance of the nervous system, a peculiar temperature record, and the very significant Widal reaction and absence of leucocytosis. What exactly produces the typhoid state it is difficult to say. Milner Fothergill[1] considers it to be without in-



creased renal activity, and with the accumulation in the blood of the products of the tissue-waste.

At times we meet with a fever attended with typhoid symptoms and diarrhœa due to contaminated drinking-water. The septic fever, of which on one occasion I saw a number of instances at the Pennsylvania Hospital among sailors from drinking bilge-water, is, however, of comparatively short duration, and has not the characteristic temperature record or eruption of typhoid fever.

Enteritis.—The great difference between enteritis and typhoid fever consists in this: in enteritis the inflammation of the intestine constitutes the disease; there are no symptoms other than those referable to the inflamed intestine. We find no great prostration; no mental wandering; no enlargement of the spleen; no rose-spots; no signs of abnormal processes due to a typhoid dyscrasia. The disorder, too, gives rise to much more abdominal pain, and is of shorter duration. In certain rare cases the follicles of the intestines are inflamed and swollen, and the attending febrile malady may closely simulate typhoid fever, without, however, its characteristic intestinal lesions, or eruption, though with considerable diarrhœa and swelling of the spleen.[1] Again, I have known fecal accumulations in the intestine to produce and keep up diarrhœa and continued fever of several weeks' duration similar to that of typhoid, and ceasing only when the large fecal masses were voided. The absence of eruption, of cerebral symptoms, and of enlargement of the spleen proved the points on which the correct diagnosis of the non-existence of typhoid fever was based. In all such cases the Widal test would be of value.

Peritonitis.—The same remarks apply to peritoneal inflammation. Here, moreover, the expression of the face, the constipation, and the great abdominal tenderness serve as marks of discrimination. The low continuous fever in tubercular peritonitis may be very misleading, as well as the gradual development of the disease and the tympanitic distention. But the history of the case, the irregularity of the fever, the supervention of ascites become very significant. On the other hand, we must not forget that acute peritonitis may appear in the course of typhoid fever. Generally this untoward event happens at a late period of the disease, and is connected with intestinal perforation, and, as a general diagnostic rule, we are right in assuming, when peritonitis is found in typhoid fever, that there has been perforation. But in very rare cases there is no such association.

Appendicitis.—The differential diagnosis between typhoid fever and

[1] Cazalis and Renaut, Archives de Physiologie, 1873.

appendicitis has been inquired into with the latter affection: their coexistence has been just mentioned with the complications of typhoid fever.

Meningitis.—Typhoid fever has some symptoms in common with inflammation of the brain; but the signs of difference have been discussed in connection with acute meningitis, and need not here be examined. The temperature record is very significant, and Kernig's sign is said to be absent.[1] But in rare cases we really have meningitis as a complication of typhoid, showing small pupils, strabismus, vomiting, and rigid neck; in the exudate in the meninges typhoid bacilli have been found. The distinction from epidemic cerebro-spinal meningitis we shall presently trace.

Ulcerative Endocarditis.—In some cases the differential diagnosis between this and typhoid fever becomes of great difficulty, especially if the case be not seen until the endocarditis have led to delirium and the symptoms of collapse. Recurring chills, with high temperature and sweats, as in malarial fever, great rapidity of pulse, with sudden changes and marked irregularity, a generally diffused roseolous eruption, and the signs of the cardiac lesion, form the most trustworthy points of distinction.

Acute Pulmonary Affections.—In the majority of cases of typhoid fever we find cough, dependent upon an affection of the bronchial tubes. The bronchial affection gives rise to extreme loudness of the râles, with a cough disproportionately slight; sometimes, too, owing to the blood gravitating to the most dependent portions of the lungs, the resonance over the posterior part of the chest is impaired. From these phenomena, added to the abdominal and cerebral symptoms of the fever, the eruption, and the vital depression, there is no difficulty in discriminating between idiopathic *bronchitis* and typhoid fever.

Not infrequently we find a dry *pleurisy* combined with the bronchitis, and in some cases, not in many, the cough is associated with exudation into the pulmonary structure. Now, it may be extremely difficult to distinguish a *pneumonia* of this kind from inflammation of the lung setting in amid signs of prostration, until the eruption and the abdominal symptoms solve the difficulty. Generally, however, it is not a matter of much doubt, as the condensation of the lung in typhoid fever does not occur early in the disease,—not, in fact, until the symptoms of the fever are clearly developed.

At times, however, typhoid fever sets in acutely with the signs of acute lobar pneumonia; there is a chill, followed by high fever; there

[1] Keller, Revue des Maladies de l'Enfance, Sept. 1898.

are no abdominal symptoms. The lung consolidation does not undergo resolution, and in the second week or later diarrhœa appears, and the characteristic eruption of typhoid fever may or may not show itself. The general typhoid condition gradually becomes marked. It is very difficult to distinguish these cases of so-called *pneumo-typhus* —chiefly described by Wagner[1] and other German observers—from pneumonia of a low type; they depend upon early and extreme bacillary infection of the lungs. The eruption, when present, is very valuable, as is the Widal reaction.

Occasionally a cough remains after the typhoid fever has left. The patient soon loses the strength he may have acquired; the temperature is again higher, and over both lungs many fine, dry, or moist sounds are heard. The percussion-note is here and there dull; profuse expectoration, dyspnœa, and excessive sweating are noticed. An examination of the sputum shows that the case has become tubercular. But, as regards the lung symptoms of typhoid fever, we must always bear in mind that acute pulmonary tuberculosis may simulate it; the high fever, the prostration, the scattered râles in the chest, with here and there spots of dulness, even the delirium, the stupor, and the enlargement of the spleen may be present; but the eruption never is, and the diarrhœa rarely. In general acute miliary tuberculosis the similarity is even greater, and diarrhœa is not uncommon; the disease is, as a rule, longer. Tubercle bacilli may or may not be present in the sputum; they have been detected in the urine and in the blood; when present they enable us to make a positive diagnosis. In rare instances the two diseases coexist.

In concluding the subject of typhoid fever it will be proper to notice those forms of the affection which run their course in a different manner from that ordinarily pursued by the malady,—there are especially two,—the *mild typhoid* and the *abortive typhoid*. The former has usually a gradual beginning, and the disease throughout remains mild; its duration may be, however, the same as, or even longer than, that of ordinary typhoid, or it may be considerably shorter,—in fact, an abortive typhoid, the variety of typhoid to which Jürgensen especially has directed attention.[2] Yet the abortive type is not always mild; cases are mentioned[3] in which the temperature rose to 106°, but in which the duration of the fever was only from seven to twelve days.

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[1] Archiv für klin. Med., Aug. 1884.

[2] Sammlung klinischer Vorträge, No. 61, 1873. See also paper by Johnston, Amer. Journ. Med. Sci., Oct. 1875.

[3] Liebermeister, in Ziemssen's Cyclopædia.

Indeed, it is the short duration that is characteristic of *abortive typhoid*. As a rule, it begins suddenly, and the temperature reaches its highest point on the second or third day. It often does not exceed 104°, and it stays at, or near, the height it has so speedily attained for the greater part of the duration of the fever, and then remissions show themselves, and there is a gradual return to a healthy standard, much in the same way as at the end of ordinary typhoid fever; or the changes are so marked and rapid that the defervescence is accomplished in a few days. The symptoms of typhoid fever are all met with in the abortive malady, though they are not present with the same constancy; tenderness in the right iliac fossa is the most frequent; enlargement of the spleen and the rose-colored spots are very usual; diarrhœa is often wanting. The disease terminates in sixteen days or less; but there is great proneness to relapses. It is not apt to be a fatal affection. I am certain it is one very rarely seen in this country.

Much has been said about *mountain fever*, especially as it has been observed in Colorado and other mountainous regions of the Western States, being a separate form of fever. But it is not; it is an irregular form of typhoid in which the eruption is often absent. The observations of Woodruff[1] and of Raymond, who got characteristic reactions with the Widal test, remove any doubt as to its nature that may have existed. Bradycardia, or slowness of pulse, is often present.[2]

Another variety of typhoid fever is occasioned by the *coexistence with malaria*. The manifestations of this occur mostly late in the disease, and chills are apt to call our attention to the character of the malady. But chills often happen from other causes in typhoid fever: from cholecystitis, from peritonitis, from appendicitis, from pyæmia, from masturbation,—of which I once saw a striking illustration,—from the decided use of antipyretics, especially the coal-tar preparations, and sometimes without discernible cause. To be sure that the chills in typhoid fever are malarial, we must find the malarial organisms. But we shall, farther on, examine the association of malaria with typhoid fever more in detail.

In conclusion, the interesting question arises, In how far can we recognize *typhoid fever without intestinal lesions?* We now know that this happens; the bacillus typhosus has been found in the gall-bladder, gall-ducts, lungs, and elsewhere, and there has been a positive Widal reaction without any other marked sign. As yet we are not in a condition to be sure of such a form of typhoid fever. There is always the possibility of a previous attack of typhoid being the cause of the

[1] Amer. Journ. Med. Sci., March, 1898.

[2] Raymond, *ibid.*

Widal reaction. But it is a question whether irregular forms of fever, with persistent slight elevations of temperature and general depression, for which no organic cause can be found, or many of the instances of afebrile typhoid fever, are not illustrations of this kind of typhoid infection.

Typhus Fever.—This is a highly contagious malady, almost always met with in an epidemic form. It prevails in jails and camps, among crowded, underfed populations, or in badly ventilated localities, and has no constant structural lesion. In this country it is a very rare disease; indeed, it is becoming rarer everywhere. It is either preceded by a brief stage of lassitude and dejection, or is ushered in with a chill and pain in the head and back. The skin soon becomes dry and of pungent heat; the pulse rises much in frequency, and is at first full, sometimes even tense. The patient lies in a state of half-consciousness, dull, drowsy, weak, with evident signs of his nervous and muscular system being overwhelmed by the influence of some fearfully depressing poison. There is headache and giddiness; the face is flushed, the eye injected; the odor from the body extremely unpleasant.

By the fifth day all these symptoms are plainly marked, and about this time a coarse, red eruption makes its appearance. But it occasions no change in the gravity of the symptoms. On the contrary, these increase; the patient wanders, picks at his bedclothes, and ceases to complain of the pain in the head or limbs. The pulse is frequent and feeble; the tongue dry and dark; sordes collect on the gums and teeth. The bowels remain as they were at the onset,—constipated. The urine often comes away drop by drop, or, as the bladder loses the power of contracting, is retained. The case has now reached its height; the signs of a prostrated nervous system, of deteriorated blood, and of utter loss of muscular strength either begin to pass away, or deepen from hour to hour and clearly show the doom that awaits the fever-stricken patient. From the beginning of the distemper until the unfortunate issue is rarely over thirteen days. If the sick man can withstand the poison until the third week, he is apt to throw it off and recover; but it may be so virulent as to overpower him almost at the onset.

Micro-organisms have been found in cases of typhus fever, though it is not certain that they are characteristic. Dubief and Bruhl[1] have found a diplococcus, chiefly in the lungs and bronchial secretions, that they designate "diplococcus exanthematicus." Andrew Balfour and

[1] Universal Medical Journal, May, 1893.

Porter[1] isolated a diplococcus not identical, detected also in the blood which retained the stain by Gram's method, and which they believe to be the specific bacillus.

Let us examine some of the symptoms of the pestilential disease:

The skin is covered with a characteristic *eruption*, from which the disease takes its name of "spotted" or "maculated" or "exanthematic" typhus. The rash is at first slightly elevated and much like that of measles. It is of a dark tint, a "mulberry rash," and fades but does not vanish on pressure. It makes its appearance from the fifth to the seventh day, and is permanent, consisting not of successive eruptions, but of the same spots, which deepen or lighten with the changes in the disease, and do not pass away before the fourteenth day. Each spot thus lasts until recovery or until death, and no new ones show themselves after the second or third day of the rash. They are numerous on the trunk and the extremities, but are rarely observed upon the face. Some are much lighter than others, and thus a mottled aspect of the skin is produced. Sometimes the spots are of purple color and uninfluenced by pressure. These petechiæ are attendants of the worst forms.

The skin of a typhus-fever patient is often sensitive, and generally very hot. In some cases the thermometer indicates a *temperature* of 107°, or more; commonly it ranges above 104°. The heat is sustained: it does not show the decided differences between morning and evening that are observed in typhoid fever, the daily variations to the middle of the second week being rarely one degree; and from that time onward the morning abatement does not amount to more than about 1.5°, until the defervescence is reached. The passing away of the high temperature occurs, however, not, as in enteric fever, by more and more evident remissions, but suddenly. Early in or towards the middle of the third week the temperature falls quickly, and in twenty-four or thirty-six hours a normal standard is reached. In rare instances, the temperature may not rise above the normal, or may be subnormal.[2]

The *cerebral* symptoms of typhus fever are never absent. Stupor is frequent. The patient lies in a heavy slumber, occasionally muttering some incoherent words; or he is sleepless, his eyes remain wide open, he has *coma-vigil*, he takes no notice of anything going on around him. Either of these states may deepen into coma. In other

[1] Edinb. Med. Journ., Feb. 1899.

[2] Combemale, Gazette hebdom. de Médecine et de Chirurgie, 1893, No. 30, p. 352.

cases delirium is the most conspicuous symptom. This delirium rarely sets in before the end of the first week. In type it is low and muttering, and unaccompanied by great restlessness; or it may be associated with constant movements and trembling of the limbs, or jerking of the tendons,—in fact, with hysterical symptoms. Sometimes the mental wandering is active and very persistent. The patient can hardly be restrained from getting out of bed. He has illusions of hearing and of sight; his eyes are injected, the pupils often contracted; there is headache, with intolerance of light. Here we have the true brain typhus, with its formidable cerebral symptoms simulating closely those of *acute meningitis*, and differing only by their union with a cutaneous eruption, by the absence of strabismus and of rigidity of the neck, by the dissimilar aspect of the tongue, the great prostration, and by the beat of the pulse, which is rarely full, and never so tense as that of meningitis. Convulsions, should they occur, are generally of uræmic origin.

The head-symptoms of typhus are, like those of enteric fever, sometimes connected with a noisy, shallow, and irregular respiration. This kind of breathing can be clearly traced to the abnormal state of the nervous system, as no signs of alteration in the lungs coexist. Often, as Flint[1] has pointed out, it is a forerunner of fatal coma. In one case I found the strange phenomenon associated with distention of the bladder, and subsiding after the introduction of a catheter.

The remarks with reference to the cerebral phenomena of typhus apply to those instances in which there is no inflammatory disorder within the cranium. But we must not overlook the fact that this may ensue. Such cases are difficult of recognition. The pulse, as a rule, is slow and irregular, the pupils are contracted, there is a frown on the forehead, and intense headache, sometimes screaming. Vomiting is not always encountered. The morbid appearances may be confined chiefly to the base of the brain.[2]

The *pulse*, after the disease is fully developed, is generally rapid, and of moderate volume or feeble. The beat of the heart may be excited and violent, while the pulse is very weak. Often the cardiac impulse undergoes a great diminution, and with its change the first sound becomes enfeebled; in fact, it is sometimes almost lost, and only very gradually regains its natural tone. Occasionally, at the height of the disease, it is replaced by a soft, systolic murmur of blood origin.

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[1] Clinical Reports on Continued Fever.

[2] Kennedy, Dublin Quarterly Journal, Feb. 1867.

The *urine* is generally high-colored at first, and deposits an abundance of urates and phosphates. There is an absence of the chlorides, or they are reduced to a trace. The urea, as ascertained by Parkes[1] in a case in which no medicine was given, is increased: during convalescence it sinks below the normal standard. In eight out of twenty-one cases that I examined during an epidemic[2] the urine contained albumin, but this ingredient was present only in the severer cases. Tube-casts, either finely granular or hyaline, or epithelial, are also found.

There is usually no *Widal reaction* in typhus fever. Harvey Littlejohn and Ker found it only twice in twenty cases, and it was not certain whether the two cases had not had previous attacks of typhoid fever.[3] Creemans[4] reports the Widal reaction as present in one case of typhus fever confirmed by autopsy.

The *complications* encountered during the course of the fever, or during convalescence, are much the same as those of typhoid fever, although they do not in the two diseases occur with equal frequency. We meet with abscesses, with large sloughs on the trunk and extremities, or with gangrene of the extremities, with milk-leg, with erysipelas, with inflammation of the parotid gland, with œdema of the glottis, and with pulmonary complaints. The latter are very common, and mostly very alarming. Sometimes they consist merely in affections of the larger or the smaller bronchial tubes, and râles of varying size are heard all over the chest. At times, instead of these signs, or associated with them, may be noticed dulness on percussion and bronchial respiration over the lower lobes of the lungs, depending upon congestion, with consolidation, more or less perfect, of the pulmonary tissue. Here is one of the worst of all the complications,—a low form of pneumonia, often of the bronchio-pneumonic type. During the last stages of typhus fever, or after convalescence has set in, gangrene of the lungs occasionally develops in the lungs, with the same symptoms as during or subsequent to typhoid fever.

To discuss now the *differential diagnosis* of typhus fever. We find various maladies resembling it, but none so closely as *typhoid fever*. The subjoined table shows both their similarities and their differences:

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[1] The Urine in Disease, p. 156.
[2] Amer. Journ. Med. Sci., Jan. 1850.
[3] Edinburgh Med. Journ., July, 1879.
[4] Transactions of the College of Phy[illegible] Philadelphia, Nov. 1880.

Typhoid.	Typhus.
Age generally from eighteen to thirty-five.	At all ages; often in persons beyond middle life.
Not contagious; mostly sporadic.	Highly contagious; usually epidemic.
Attack generally insidious.	Attack generally sudden.
Duration fully three weeks; frequently much longer.	Duration somewhat shorter; often not prolonged beyond second week.
Death hardly ever before end of second week; more generally in, or after, third week.	Death not infrequently at end of first week, and often before conclusion of second.
Cerebral symptoms come on gradually; last longer.	Delirium or decided stupor comes on soon, sometimes almost from the onset; headache has appeared and disappeared by about the tenth day.
Great emaciation.	Less emaciation; greater prostration.
Face pale, or flush confined to cheeks.	Face deeply flushed; eye injected.
Characteristic temperature-record, chiefly influenced by the changes in the intestinal glandular lesion.	Temperature-record more that of a continuous fever; for the most part sudden and rapid defervescence.
Abdominal symptoms, such as diarrhœa, tympanites; stools contain characteristic bacilli; intestinal hemorrhage not unusual.	No abdominal symptoms; bowels constipated; meteorism rare; intestinal hemorrhage of extreme rarity; sometimes acute dysentery during convalescence or as a sequel.
Epistaxis common.	No epistaxis.
Bronchitis aud pleurisy; pulmonary congestion.	Intense pulmonary congestion; bronchopneumonia.
Eruption light red in fine spots, and not on extremities or face.	Eruption darker in color, coarser, and all over body; seldom on face.
Widal test positive.	Widal test generally negative.

Yet it is occasionally very difficult to come to a conclusion between typhoid and typhus fever, on account of the measly rash that the former exceptionally presents; or the symptoms of the two diseases are strangely blended or interchanged. Thus, we may have constipation in typhoid, and diarrhœa in typhus, or the eruption may be curiously mixed. For instance:

A boy, sixteen years of age, was received into the Philadelphia Hospital, with evident signs of a beginning fever of a low type. A day or two after his admission, and corresponding, as nearly as could be ascertained, to the fifth day of the disease, an eruption showed itself all over the body. It was dark-colored, petechial in its aspect, and did not disappear on pressure. Associated with it were drowsiness and constipation. In a few days more, however, the symptoms changed. The dark eruption faded, and rose-colored spots were perceptible on the chest and abdomen; diarrhœa set in, and the fever

ran its course to a favorable termination with the character of typhoid, just as at the onset it had assumed the character of typhus.

Besides typhoid fever, typhus may be confounded with meningitis, with inflammation of the lungs, with measles, with smallpox, and with the plague. The distinctive marks between the first two and typhus fever have been rendered apparent while discussing the cerebral and pulmonary complications of the malady. I shall here only dwell again upon the great value of the eruption from a diagnostic point of view. The symptoms that approximate measles, smallpox, yellow fever, cerebro-spinal fever, and the plague to typhus will be analyzed in connection with these affections.

Cerebro-Spinal Fever.—This disease is also known as cerebro-spinal typhus, as epidemic meningitis, and as epidemic cerebro-spinal meningitis, and is the affection which has been called in this country spotted fever. It was formerly very prevalent in portions of the United States, but the present generation of physicians had little knowledge of it until about simultaneously with the severe epidemic in Germany in 1863 and 1864 it invaded this country and committed great ravages, especially in some of the New-England States, in New York, and in Pennsylvania. Since that time it has become naturalized here, as Ziemssen states to be also the case in Germany.[1] There was an epidemic in Boston in 1897 and 1898, and I saw a number of cases in Philadelphia in the early spring of 1899.

Cerebro-spinal meningitis does not always present exactly the same symptoms. These vary somewhat according to the structures which bear the brunt of the disease. Usually, however, marked cerebro-spinal phenomena preponderate; in some instances the evidences of pulmonary embarrassment or of blood deterioration are very prominent. Again, the signs of spinal disturbance may prevail over those of the cerebral, or the reverse.

The disease may be gradual in its approach, feelings of chilliness, succeeded by headache, by tenderness at the nape of the neck, by nausea, and by pain in the back and joints, preceding its full development. Generally its onset is sudden; a violent chill is quickly followed by intense headache, vomiting, and extreme prostration. However the beginning, the disease usually soon reaches its full development. The excruciating headache is associated with vertigo, and often with delirium and stupor. The headache may remit, but does not cease during the attack. Another symptom of the fully developed disease is stiffness of the deep muscles of the neck, so that the patient

[1] Cyclopædia of the Practice of Medicine, vol. ii., 1875.

cannot bend the head forward; and the stiffness may pass into marked contraction, and the head be thrown backward and rigidly fixed. The contraction of the muscles may extend along the spine, which frequently is painful, not specially to the touch, but on movement of any kind; sometimes, moreover, severe spontaneous pain occurs. There are also pain at the nape of the neck, and in the loins and shooting to the lower extremities, and pain at the epigastrium, and a feeling of contraction of the chest. The Kernig sign of meningitis is always present. The face has a fixed or suffering expression; the patient is extremely restless; he trembles; talks incoherently; when spoken to, does not appear to hear; his pupils are contracted or dilated and often unequal, and there may be dimness of sight, or double vision and strabismus. The skin is dry, generally very sensitive, or in some parts the sensibility is increased, in others diminished, and the cutaneous surface is frequently spotted with a red eruption, erythematous and roseolous,—an eruption which often becomes brownish, and then for the most part rapidly petechial, and is wholly uninfluenced by pressure; or the purple spots may be seen from the start. Vesicles, too, commonly appear on the lips. They show themselves from the third to the sixth day of the disease, while the eruption is seen on the first day, or may at all events be detected by the third day. The blood rapidly undergoes changes. I have found marked blood-murmurs in the heart in a case of but two days' duration.

The pulse at first is natural or slow; but it becomes rather frequent and irregular, and commonly remains accelerated throughout the disease, showing extraordinary variations in a few hours; the impulse of the heart is at times much augmented. The tongue is moist or dry, and brown; the breathing often hurried and shallow; and the urine I have often noticed to contain large quantities of urates and to be slightly albuminous; hyaline and granular tube-casts are also found in severe cases; in the malignant cases there may be hæmaturia. The bowels are at the outset constipated; but as the malady advances they may become relaxed; in some cases dysentery has been observed. There are usually persistent irritability of the stomach, with great thirst, and spasmodic contractions or convulsive movements in the muscles of the extremities. The spleen, early in the affection, is apt to enlarge, but does not continue tumefied. With these symptoms, to which those of exhaustion become plainly added, the disorder progresses to its close, presenting now and then strange and delusive remissions, soon followed by distinct exacerbations. In fortunate instances the morbid phenomena gradually lose their vio-

lence, and the patient, greatly emaciated, enters upon a tedious convalescence.

But though these are the symptoms which frequently recur in epidemics, yet as already indicated, they cannot always be taken as the standard expression of the disease. The temperature is most variable; it may be scarcely above the norm, or may reach between 106° and 108°, or even higher, without there being a proportionate rise in the pulse. Irregularity of the temperature is a very common and significant feature. High temperature may be interrupted by long-continued normal temperature, and sometimes the type of fever is like that of a tertian intermittent, but with much longer paroxysms.

In an epidemic in a mining centre in the State of Maryland, carefully investigated by Flexner and Barker,[1] symptoms referable to the cranial nerves were especially observed, particularly loss of smell, strabismus, nystagmus, inequality of the pupils, photophobia, ptosis, impairment of vision, deafness, rigidity of the face, trismus, besides Cheyne-Stokes breathing and disturbances of speech. The strabismus was divergent, and in many cases affected especially the right eye.. A considerable number presented engorgement of the retinal veins; some, optic neuritis. The tendon-reflexes were not uniform, but were in many cases diminished. In addition to herpes and purpuric and petechial spots, a common form of cutaneous eruption was an indistinct purplish mottling of the surface. Nearly twenty per cent. of the cases presented articular complications, principally effusions into and around the joints, with redness and swelling. Well-marked leucocytosis was a constant feature at the height of the disease; the red blood-corpuscles were little, if at all, changed in number, while the hæmoglobin was somewhat diminished. Leucocytosis was also observed in every instance of the disease seen by Osler[2] in an outbreak in Baltimore in 1898.

The duration of the malady is very various. Patients may become rapidly comatose, and die within twelve hours, before any distinctly febrile action has begun; or may sink in a few days; or, on the other hand, the complaint may pursue a chronic course, lasting for many weeks, and during this time deafness and blindness, convulsions, retention of urine, and local palsies—though these are unusual —may be prominent phenomena.

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[1] American Journal of the Medical Sciences, Feb. March, 1894. For a report of the ocular findings, see, also, Randolph, Bulletin of the Johns Hopkins Hospital, 1893, vol. iv., No. 32, p. 59.

[2] Cavendish Lecture, Phila. Med. Journ., July, 1899.

The cause of epidemic cerebro-spinal meningitis is the diplococcus intracellularis meningitidis of Weichselbaum, also called the meningococcus. It is often found in association with the pneumococcus, and, indeed, the frequent clinical combination with pneumonia shows a close connection between the two micro-organisms.

A valuable means of diagnosis of cerebro-spinal fever has been found in lumbar puncture introduced by Quincke. The puncture is best made between the second and third, or the third and fourth, lumbar vertebræ with an ordinary exploratory needle, local anæsthesia by

FIG. 77.

The diplococcus intracellularis, obtained from a lumbar puncture of a case of cerebro-spinal meningitis at the Pennsylvania Hospital, by the pathologist, Dr. Cattell.

a freezing mixture having been previously produced. After the needle has been passed to about four centimetres in children, and double this distance in adults, the fluid generally comes out drop by drop, and from five to ten cubic centimetres should be collected in a completely sterilized culture-tube, which is then tightly plugged. The fluid may be clear or turbid; in severe cases it is usually turbid. Bacteriologically studied, it is found to contain the organisms always detected in cerebro-spinal fever, and the earlier in the case the lumbar puncture is made the greater is the chance of finding the diplococcus intracellularis. No evil effects follow from lumbar puncture. Williams found it even beneficial to the patient, an opinion which Wentworth does not share, believing any relief to be but for a

few hours. Weichselbaum's method is very generally followed, and was made use of by Councilman, Mallory, and Wright in their valuable study of epidemic cerebro-spinal meningitis.[1]

Cerebro-spinal meningitis attacks children frequently. It is more common in winter and in spring than in summer; though I have seen it in summer. It is an affection familiar to military surgeons: it arises at periods when troops have been subjected to unaccustomed fatigue or have been huddled together in unhealthy barracks or camps.

To determine the diagnosis is ordinarily not difficult: the sudden onset of the malady and its epidemic character are safeguards against error. The protracted cases simulate *typhoid fever*. They resemble it in its long duration, in several of the cerebral symptoms, and in the occurrence of an eruption and sometimes of diarrhœa. They differ from it in the more abrupt invasion, in rather in the short time in which the disease reaches an alarming aspect; and, in the early stages, the violent headache, the nausea, the constant vomiting, the slow or normal pulse, and the temperature, are unlike the signs of enteric fever. In those cases in which an eruption appears, it is noticed, at least, by the third or fourth day, not at the end of a week as in typhoid fever; nor is the rash, save in extremely rare instances, rose-colored. The cerebro-spinal form of both typhoid and typhus fever is often mistaken for cerebro-spinal fever, and there may be, indeed, much similarity in the cerebro-spinal symptoms.[2] But the eruptions of these fevers are of great diagnostic value, as is the enlargement of the spleen, and they do not present the fixed spinal pain, the severe muscular twitchings, the great stiffness of the muscles of the neck and rigidity of the muscles of the back, the labial herpes, the irregular temperatures we find in epidemic cerebro-spinal meningitis. Then the Widal reaction in typhoid fever, and the lumbar puncture in cerebro-spinal fever, would give most valuable evidence.

The suddenness with which the morbid phenomena occasionally develop, and the falls that take place in the course of the affection, may cause it to be mistaken for the cerebral variety of *congestive fever*. But the remissions are not so marked as in this pernicious malady, nor are the exacerbations preceded by a long, violent chill. Moreover, the temperature-record is different, and congestive fever does

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[1] Councilman, Mallory, and Wright's "Epidemic Cerebro-spinal Meningitis."

[2] Report of the State Board of Health of Massachusetts, Boston, 1885.

[3] An abstract of the author's researches on this subject will be found in a series of papers now published in the American Journal of the Medical Sciences, for 1891.

not begin with congestive symptoms, but the first attack is like that of an ordinary intermittent or remittent: hence we have the history of the case to instruct us. Finally the detection of hæmatozoa in the blood establishes the diagnosis of the malarial affection.

From *tetanus* cerebro-spinal meningitis may be distinguished by its epidemic prevalence, and by the signs of mental disturbance, which are very slight or wholly wanting in the former disorder. Trismus is common and early in tetanus; very rare in cerebro-spinal fever. Generally, too, the sudden and painful spasms, aggravating the tetanoid contractions, and the cognizance of the exciting cause of the tetanic convulsions, such as their following wounds or punctures, aid in interpreting their meaning.

How can we discriminate between *inflammation of the meninges of the cord* and epidemic cerebro-spinal meningitis when protracted? By the history of the case, the mental symptoms of the cerebro-spinal fever, the eruption, and the persistent rigidity of the muscles, rather than the clonic spasm so much more common in the former malady.

Tubercular meningitis is distinguished by its insidious beginning, the generally much more protracted course, the absence of eruption, and usually of marked stiffness of the neck, the variations in the pulse according to the stage of the malady, the irregular breathing, and the history of a tubercular taint.

Idiopathic or *sporadic cerebro-spinal meningitis* is a very rare disease. It runs a much slower course than the epidemic malady generally does, and its spinal symptoms are less marked. But it cannot be distinguished with any certainty from sporadic cases of cerebro-spinal fever. The absence of an eruption and of the striking variations of temperature presented by the latter is of significance. But as the diplococcus intracellularis has been found in the sporadic cases,[1] these represent the same disease as epidemic cerebro-spinal fever, only in a somewhat dissimilar form. It is, indeed, a question whether there are not yet other forms due to this, for in typical anterior poliomyelitis the same organism has been found by lumbar puncture.[2]

As regards the different forms of ordinary meningitis, the distinction, except by laying stress on the epidemic character of the disease, is not easy. The eruption is wanting in these, and the spinal symptoms are far less pronounced. The history of the case, too, is important, as pointing to blow or injury, to syphilis, to extension of disease

[1] Still, Journal of Pathology and Bacteriology, vol. v., 1898.

[2] Schultze, Münchener Med. Wochenschr., 1899.

[illegible]

In some instances of cerebro-spinal fever there is great pain, with some swelling of the joints, and the disorder is thought to be *acute rheumatism*. But the head-symptoms, the state of the muscles of the neck, and the dissimilar course of the malady soon clear up the diagnosis.

The poison may produce so light a case that the stiffness of the neck may be mistaken for *rheumatism of the cervical muscles*. There is, however, even in these instances, an unusual amount of headache, and in a case in which I was consulted it became a permanent condition for several years, and then yielded.

Uræmia with contracted kidneys may give us most of the same symptoms as cerebro-spinal fever, especially headache, vomiting, and retraction of the head; the differentiation will depend upon the previous history, the presence or absence of febrile phenomena and of cutaneous eruptions, and an accurate examination of the urine.

From the cerebral form of *typhus*, the dusky countenance of the latter, the characteristic eruption, the regularity of the high fever, the violent delirium, and the absence of marked spinal symptoms, distinguish epidemic cerebro-spinal meningitis. Most of the same symptoms differentiate it from the cerebral form of *typhoid*, and, in addition, we have, as the case progresses, the important aid of the Widal test.

Cerebro-spinal fever may, during an epidemic, complicate other acute maladies, and mix its symptoms curiously with them. With the attack the difficulty does not pass off, for it may leave want of power and all kinds of local palsies, besides derangement of vision, permanent deafness, impaired intelligence, epilepsy, persistent headache, chronic meningitis, which may be indeed the cause of the headache, and chronic hydrocephalus. In one instance I have known an extraordinary swelling of the whole body to follow; the skin is hard, tense, and greatly thickened, pits very little on pressure, except around the ankles, and is tightly drawn over the face; this swelling and thickening, very much like a general sclerema, has now lasted for upward of twenty years, and has been attended with a feeling of numbness in the skin and a moderate amount of anæmia. There is no palsy or albuminuria; the patient suffers little inconvenience, except from her size. She has a waxy countenance, and looks like a very fat woman.

Relapsing Fever.—This is a form of fever characterized by its rapid course and its proneness to relapse. Epidemics of this disease —and it occurs only in epidemics—are frequently encountered in Ireland and in Scotland. There was an epidemic of it in New York and in Philadelphia in 1869.

The disorder is decidedly acute. Its invasion is sudden, and marked by rigors, pain in the back and limbs, vertigo, severe headache, and nausea and vomiting. Fever is soon developed, and rises high, to from 104° to 107°. There are severe muscular pains, particularly in the muscles of the extremities; the pulse is very rapid; the temporal arteries throb; the tongue is covered with a thick white fur. The bowels, as a rule, are constipated. In many cases there is engorgement of the liver, with yellowness of skin; and in nearly all there are epigastric tenderness and marked enlargement of the spleen. The matter ejected from the stomach is greenish, or sometimes black and like coffee-grounds. Minute points of extravasated blood are not uncommonly seen upon the integument. The urine is scanty, and contains usually bile-pigment, some albumin, and hyaline casts. On the fifth or the seventh day, though sometimes not until the tenth, the symptoms subside as speedily as they set in, a profuse perspiration preceding their decided abatement, and the temperature falls to the norm or even below. Convalescence is now apt to be rapid, and seemingly complete, the patient being up and going about; but the apparent return to health does not last long. Ordinarily after a week, therefore on the twelfth or fourteenth day from the beginning,—sometimes sooner, rarely later,—the attack, preceded perhaps by a slight rise in temperature for an evening or two, returns, presenting again the same signs, and again terminating by a critical sweat in convalescence. This second attack may be short and mild; but it may be both longer and of graver character than the first. It is, at times, followed by another, and yet another, relapse. When the patient finally throws off the disease, he is very weak, and his blood is much impoverished. He shows a tendency to dropsy of the extremities; and blowing murmurs, evidently not organic, are perceptible while listening to the heart. These murmurs, however, may also be heard during the paroxysms. The patient is not really well during the intermission; his spleen remains enlarged, the pulse is slow, the action of the heart is weak, and the muscular and arthritic pains do not entirely disappear.

Relapsing fever has an intimate connection with destitution. It is a contagious but far from a fatal disorder, except, perhaps, in the negro. In fatal cases death sometimes happens during the first paroxysm as the result of syncope, of hemorrhage into the brain or from the lungs; or it may occur suddenly during the intermission from paralysis of the heart. But the most common termination of the cases having an unfavorable issue is in consequence of complications or of states which have been induced by the malady, such as

lobular or lobar inflammation of the lung, hemorrhagic pachymeningitis, abscess of the spleen or of the kidney leading to pyæmia, Bright's disease, dropsy, chronic diarrhœa, parotitis, palsies. At times the patient perishes in a condition similar to the collapse of cholera, though the collapse is more protracted and the pulse can be felt, and discharges from the bowels are by no means a constant accompaniment. The extreme prostration, attended with great coldness of the skin, may last for days. It is more particularly met with in the "bilious" or "bilious typhoid" form of the malady,—a dangerous variety, in which severe vomiting, jaundice, and delirium are encountered, and the paroxysm is not followed by a distinct intermission or remission, but often by the signs of collapse mentioned, in which uræmic symptoms have been more particularly noticed.[1] The collapse, however, may happen not only at the close of the paroxysm, but in the remission, whether this be distinct or not, or in a subsequent paroxysm; and this may be the case no matter what variety of the disorder we have to deal with, and whether or not the serious symptoms be due to uræmia.

Yet the state of the kidneys and of the urinary secretion has commonly much to do with the graver phenomena of the malady. Actual renal disease with albumin and tube-casts in the urine was discerned by Obermeier[2] in two-thirds of his cases. It was also, with or without tube-casts, met with in a number of Pepper's cases.[3] The urea is increased and may be retained, thus occasioning grave symptoms. Leucine and tyrosine have been also found.

There is no constant obvious lesion in relapsing fever, unless it be the lesion in the spleen. This organ is greatly enlarged, and presents numerous round or irregularly shaped bodies, of white or yellowish-white color.[4] But myriads of minute organisms, spirilla, are found in the blood just prior to the outbreak of the paroxysms, and at its height. Indeed, since Obermeier's discovery of the spirilla in relapsing fever, there is no doubt that they are the cause of the malady, and their detection in the blood makes the diagnosis clear. In a single field of the microscope we may see only a few, or from twenty to thirty spirilla.

The diagnosis of the malady cannot be made positively during the primary seizure. Yet, while an epidemic prevails, it may be suspected

[1] Hermann, Account of St. Petersburg Epidemic, Schmidt's Jahrb., No. 6, 1865. See also further observations in Meissner's article, *ibid.*, No. 2, 1870.

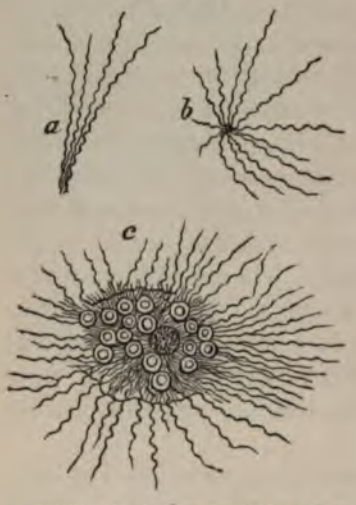
[2] Virchow's Archiv, 1869, Bd. xlvii.

[3] American System of Medicine, article "Relapsing Fever."

[4] Pastau, *ibid.*

from the fierce beginning of the attack; from the fact of the high fever-heat showing itself in less than twenty-four hours, and exhibiting either a morning remission of one to two degrees and the maximum of temperature in the early afternoon or evening, or but little difference between morning and evening, until the rapid and great fall which takes place at the crisis; and from the character of the gastric symptoms. Then the microscopical examination of the blood is of great importance. Relapsing fever resembles *yellow fever* in its short duration and in some of its manifestations. But there is this evident difference: in yellow fever the paroxysm or febrile stage is usually much shorter; the symptoms in the remission do not subside nearly so completely; this stage is a brief one as compared with the decided intermission of relapsing fever; the black vomit of yellow fever does not come on until the stage of collapse is reached; and this far more fatal malady presents lesions in the liver and heart that are not found in relapsing fever, while it does not show the extraordinary enlargement of the spleen.

FIG. 78.



Spirilla of relapsing fever (from Heydenreich): *a*, single spirillum; *b*, star-shaped bundle; *c*, nidus of spirilla, with blood-corpuscles.

From *typhoid and typhus fevers*, relapsing fever may be distinguished by the shorter prodromata, by the presence of jaundice, by the absence of the characteristic eruptions, and by the short period during which the symptoms last. Again, critical sweats with the rapid cessation of the fever are not likely to be seen in these disorders, certainly not in typhoid fever; and the very high temperature, the severe muscular and arthritic pains, the tenderness over the liver and the spleen, the vertigo, and in some cases the early collapse without apparent cause, are characteristic; while, on the other hand, delirium and stupor are rarely encountered in relapsing fever. After the relapse has taken place, the diagnosis is easy, if the case have been watched during the first attack. But, should it not have been under notice before, it may be at times very difficult, without an examination of the blood for spirilla, to say whether we are dealing with relapsing fever or with a relapse of typhoid or typhus fever. And this difficulty is enhanced by the want of uniformity of the symptoms

in the second onset of the strangely recurring malady. Another difficulty is presented by the fact that relapsing fever may exhaust itself in the first paroxysm. But this is an unusual occurrence, and the abortive cases are light. In them, too, the spirilla may be detected in the blood.

Yellow Fever.—This formidable malady takes its familiar appellation of yellow fever from the yellow tinge assumed during its course by the skin. It is a distemper met with in hot climates in low and level localities on the sea-coast. Its source is unknown; it is not malaria, nor has a characteristic micro-organism as yet been detected.[1] All we know certainly of the cause is, that the malady is due to a specific poison which does not exist without a high temperature, and that frost is its greatest enemy.

Yellow fever is an affection of short duration: it rarely lasts a week; many die on the third or the fifth day of the disease. It has but one paroxysm, which is never repeated. This paroxysm may be divided into three stages, which are well marked in some epidemics, far less so in others.

The first stage is pre-eminently the febrile stage. Its average duration is from thirty-six to forty-eight hours, but it may last three days or longer. It usually begins suddenly, and is frequently ushered in by a chill. In rare instances this is protracted, there is great internal congestion, and death ensues before reaction occurs. But much more generally a short chill is followed by decided fever. The skin is harsh and hot; the pulse quick and tense, although sometimes it is both easily compressible and not much accelerated; indeed, as a rule, it falls before the temperature declines, and there is a marked disproportion between the two. On the evening of the third day, and while the patient is still in the paroxysm of the fever, there may be, as Faget has pointed out, a temperature of between 103° and 104°, with a pulse from 70 to 80. The face is flushed; the eye brilliantly injected, yet watery. The patient is conscious, restless, anxious, and complains much of the torturing pains in his forehead, loins, and legs; the muscles of the extremities are sore when moved. The breathing is hurried; the stomach irritable, the epigastrium painful on pressure; there is great thirst. The bowels are constipated; the stools very dark-colored. The tongue is more or less coated and moist; sometimes it is red, while at other times it remains natural

[1] The bacillus found by Sternberg and called by him bacillus X is most likely the specific agent. It is very similar to the one described by Sanarelli as the bacillus icteroides. But our knowledge is as yet not positive.

throughout the disease. There is albuminuria, which, indeed, as Guitéras mentions, may be sometimes detected in the evening of the first day, and is almost always found by the third. The febrile signs increase towards evening and lessen towards morning, but do not distinctly remit until after from thirty-six to forty-eight hours, or a day or two later, when a remission does occur, or when, to speak more correctly, the whole aspect of the case changes.

The disorder now appears in its second stage, that of *calm;* the fever subsides; the pulse falls and becomes easily compressible; the headache is relieved; the breathing is no longer oppressed; the temperature declines to a little above the norm. But the gastric irritability does not wholly disappear, and a deep yellow or orange hue, which may have shown itself slightly almost from the beginning, gradually tinges the eye and the whole surface of the body. The patient is cheerful, and wishes to get out of bed. His sufferings may be, indeed, over; convalescence may have set in: after a few dark, biliary stools, the yellowness of the skin fades, and he slowly gets well.

But it is not often that the disease relaxes its hold so easily: more generally the deceptive improvement does not last a day, and after a brief lull the struggle for life begins. The patient grows again very uncomfortable and anxious, the fever rises; this secondary fever may last from one to three days, in favorable cases passing off gradually. But in severer cases, during its course, the symptoms of the first stage reappear with increased intensity. New signs, of the gravest import, show themselves; some of which are clearly due to the corruption of the blood that the poison has silently effected. The pulse sinks, and becomes slow and extremely irregular and compressible: the skin is cool, dry, dark, and in some cases of a bronze hue, or livid, and spots may be occasionally seen on its surface. The stomach is as irritable as before, but the act of vomiting is easier; and, without much retching, large quantities of altered blood, or "black vomit," are ejected. Blood oozes from the mouth, from the gums: sometimes from the eyes and nostrils, from the bowels, and from the vagina;[1] or hemorrhage takes place into internal cavities, and the blood is retained.[2]

The phenomena of collapse become now more and more unmistakable: the black vomit often ceases, because the contractile power

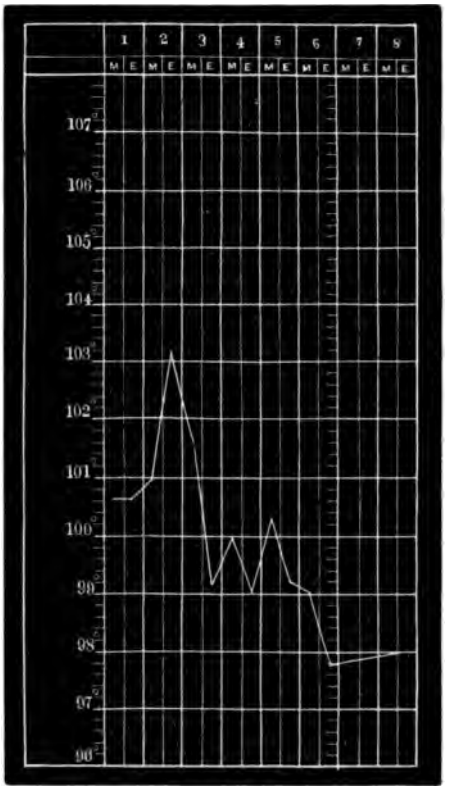
[1] Cases in the epidemic of 1856–57 at Lisbon, reported upon by Lyons. Lisbon, 1858; also by Alvarenga, Fièvre jaune à Lisbonne, Paris, 1861.

[2] In a case at the Pennsylvania Hospital the pericardium was filled with blood resembling black vomit.

of the stomach has ceased; a low, muttering delirium sets in; at times uræmic symptoms show themselves. Yet the mind may remain clear almost to the last, and the strength be but little impaired. Should reaction take place, recovery is only very gradual.

But yellow fever does not at all times and in all localities present precisely the same degree of intensity or the same group of symptoms. Sometimes it exhibits frank, active febrile phenomena; at other times there is little febrile excitement, but a disposition to internal congestions and to early prostration. This congestive form is far more dangerous than the inflammatory. Yet both are highly destructive. From 10 up to 75 per cent. are the figures representing the mortality of this fearful malady. Omitting the instances of an exceptionally mild type, the average is calculated, in the elaborate work of La Roche,[1] to be 1 in 2.32. The more rapidly the stages succeed one another, the more dangerous the case. The occurrence of black vomit, of great epigastric tenderness, of hiccough, of suppression of urine, of delirium, of early marked jaundice, of oppression in breathing, of convulsions, of a fiery, glistening eye, and of petechiæ, warrants an unfavorable prognosis. "Walking cases," or those in which the patients walk about until they suddenly eject black vomit, always terminate fatally.

FIG. 79.



Temperature of yellow fever in a case ending in recovery recorded by Bemiss.

As regards the temperature in yellow fever, the maximum elevation is attained upon the first, second, and third days of the disease, ranging from 102° to 110°; it then falls in the stage of calm, to rise usually again in the stage of secondary fever and of collapse, though it

[1] Yellow Fever, Philadelphia, 1855.

never attains the high temperature characteristic of the first stage, and never rises so rapidly. The elevated temperature of the first days may, however, continue with little variation until the sixth day, when the remission becomes marked. A complete remission usually happens on the morning of the third day, but may not occur until the fifth or the ninth. Whenever it takes place, the speedy defervescence is very characteristic. Slight rises in temperature are neither uncommon nor grave after the marked fall in the second stage. But when the temperature rises rapidly in the stage of calm it is of most serious meaning. In this stage of calm the absence of fever may be complete; but generally the defervescence is only partial: a remission, therefore, rather than an intermission.[1]

Yellow fever has rarely any complications. It may, however, seize upon those affected with other diseases. It has been specially noticed that it is frequently intercurrent in surgical and obstetrical cases.[2]

The recognition of yellow fever is, generally speaking, easy. The intense pain in the back, limbs, and forehead; the look of the face; the appearance of the eye; the color of the skin; the short duration of the high fever; the falling of the pulse while the temperature remains elevated; the nausea; the epigastric tenderness; the early albuminuria.—constitute a group of symptoms which unmistakably mark the disease.

But let us look at the points of contrast which yellow fever presents to other affections. It differs from *plague* by the absence of buboes and of carbuncles, and by the much more frequent occurrence, on the other hand, of jaundice and black vomit. Then, too, the red, suffused eye and the single paroxysm are not witnessed in plague. The lines of demarcation between the ordinary forms of continued fever and yellow fever are broadly drawn. It is distinguished from *relapsing* fever by the different countenance, by the supraorbital pain, by the early remission, and, above all, by the extreme rarity of a relapse and the infinitely greater mortality. To *typhoid fever* it bears so slight a resemblance that it is scarcely possible to confound the two affections; one, a short, severe disease, with its

[1] See on the temperature and other symptoms Faget, New Orleans Med. and Surg. Journ., 1873–74; Bemiss, Amer. Journ. Med. Sci., April, 1880, and article "Yellow Fever" in Syst. of Pract. Med. by American Authors; the temperature charts of Naegeli, of Rio Janeiro, as given by Jaccoud, Pathologie interne; Guitéras, article "Yellow Fever" in Keating's "Cyclopædia of Diseases of Children" and elsewhere; Sternberg, article "Yellow Fever" in Loomis and Thompson's System of Practical Medicine, 1897, vol. i.

[2] S. M. Bemiss, Clinical Study of Yellow Fever, *loc. cit.*

peculiar physiognomy and gastric symptoms; the other, a long-continued malady, of low type, with its characteristic eruption and enteric signs. It is only when yellow fever is protracted beyond the ninth day that the diagnosis is rendered doubtful; and then we have generally the history to guide to a correct understanding. The likeness between yellow fever and *typhus* is much closer. But one is a short fever, with distinct stages; the other is a longer, much more continued fever. One has no marked cerebral symptoms; in the other the cerebral symptoms are the most prominent feature. One has but rarely an eruption, but often hemorrhages; the other has always an eruption, and hardly ever hemorrhages.

The disease most likely to be confounded with yellow fever is *remittent fever*. In truth, the symptoms are very similar, and many of them differ only in intensity. The diagnosis of the milder forms of yellow fever from remittent fever is, indeed, extremely difficult, unless the epidemic influences prevailing be taken into account. Then, as is well known, the affections may be blended, and yellow fever become obviously periodical in its febrile phenomena. If there be coexisting malaria, we may find the malarial parasites in the blood, and we are thus deprived of the most positive means of distinction between the two diseases. Under ordinary circumstances, the detection of these, and they are generally of the æstivo-autumnal form, is of the greatest value in diagnosis. The occurrence of black vomit is not in itself a distinctive sign in yellow fever, for black vomit may be absent in yellow fever, and, on the other hand, it may, although it rarely does, occur in remittent fever, just as it has been known to occur in child-bed fever, in the plague, and even in typhus.[1] A valuable sign is derived from an examination of the urine; there is early and marked albuminuria in yellow fever.

When yellow fever is well marked, it differs in this way from bilious remittent:

Yellow Fever.	Remittent Fever.
Of short duration, ending commonly in from three to seven days.	Lasts nine days or upward.
Period of incubation from five to nine days.	Period of incubation very variable; may extend to months.

[1] This statement with reference to typhus fever is made on the authority of Stokes. The occasional occurrence of black vomit in remittent fever is admitted by many others. Some winters ago, a physician of this city brought to me, for examination, a specimen of black vomit which had the same microscopical characters that I have repeatedly found in the black vomit of yellow fever. The patient undoubtedly had remittent fever, from which he recovered.

Yellow Fever.	Remittent Fever.
A disease of one paroxysm, terminating in recovery or collapse.	A disease of several paroxysms, with intervening remissions.
Very severe nausea and vomiting throughout; early jaundice; early and decided epigastric tenderness; black vomit.	Nausea and vomiting not so severe, and rarely as marked at the onset; neither as early nor as constant; jaundice and epigastric tenderness; vomiting of bile.
Hemorrhages from gums and various parts of the body.	No hemorrhagic tendency.
Tongue clean, or but slightly coated; pulse very variable, frequently becomes slow, out of proportion to temperature.	Tongue heavily coated; pulse varies less, is always rapid until convalescence sets in.
Highly injected, humid eyes; often fierce or anxious expression of face.	Eye not peculiar; different physiognomy.
Supraorbital pain, and pain in back and in calves of the legs.	Headache; sense of fulness in head; often no pain in loins or in legs.
Very rarely delirium; mind usually clear.	Delirium frequent; mind always dull.
Urine acid, very generally contains albumin, also epithelial and granular casts and blood-casts; suppression of urine common; no micro-organism in blood; hæmoglobin in blood-serum.	No albumin in urine; suppression of urine rare; malarial parasites in blood.
Little muscular prostration; often rapid convalescence; no sequelæ.	Much greater muscular prostration; slow convalescence and tedious sequelæ.
Almost certain immunity after one attack.	One attack seems rather to predispose to others.
Very high mortality; disease is epidemic.	Slight mortality; disease more endemic in its nature.
Treatment unsatisfactory.	Very amenable to treatment.
Autopsy shows inflammation or great congestion of stomach, and some softening. Spleen slightly or not at all enlarged. Liver of a yellowish color, its secreting cells filled with oil-globules. Kidneys swollen, inflamed. Heart often exhibits granular or fatty disintegration of muscular fibres.	Autopsy shows congestion of stomach; more rarely inflammation. Markedly enlarged spleen. Liver of an olive or bronze hue, not fatty; accumulation of animal starch in liver of malarial fever, no grape-sugar.[1] Kidneys unchanged, or simply congested.

The diagnosis from *dengue*, at times a very difficult one, will be considered with this disease.

Dengue.—This is an arthritic fever with a cutaneous eruption. It is prevalent in the form of epidemics chiefly in India, and in the West Indies, in Virginia, South Carolina, Texas, and other of the Southern States. We owe some of its best descriptions to Dickson.

[1] Joseph Jones, Medical and Surgical Memoirs, vol. ii., New Orleans, 1887.

It has a period of incubation of from three to five days. It usually begins with pain, stiffness, and swelling of some of the smaller joints, or with severe muscular pains, aching in the back, and stiffness of the muscles of the neck. Fever follows, with suffusion of the face, violent headache, hurried breathing, and coated tongue; but, as a rule, without nausea and vomiting. The temperature usually attains its height, which may be 106° or 107°, within the first twenty-four hours, and then shows during defervescence marked remissions and exacerbations. On the third day the fever ceases altogether or subsides markedly, though the muscular and arthritic pains do not pass off entirely. The febrile paroxysm may last somewhat longer, indeed, five to seven days, or only six to twelve hours. In any case it is apt to be succeeded by an interval of two to four days free from absolute suffering, though not from great debility. Then the pain returns, and with it a modèrate fever; nausea and vomiting and a thickly-coated tongue, too, are noticed. This new phase of the complaint is generally relieved by the appearance of an eruption, which may be accompanied by a slight rise in temperature. The eruption shows itself on the fifth, sixth, or seventh day of the malady, and, therefore, very much later than the rash of scarlatina, which it resembles in hue and aspect. But not invariably; for it may occur in patches and be papular, or even vesicular, or like urticaria. The eruption is attended with a sense of burning and of itching, and disappears after two or three days' duration, with desquamation. It is much more pronounced than the slight and inconstant erythematous rash of the period of invasion, which disappears without desquamation with the febrile stage.

With the occurrence of desquamation following the marked rash of the third period of the disease convalescence sets in, marked by considerable muscular weakness and general depression, and frequently with the rheumatic stiffness or soreness persisting for some time. Swellings of the lymphatic glands of the neck, axilla, and groin occur in many instances, and may continue during convalescence, which in any case is apt to be prolonged, and may be interrupted by a relapse.

The cause of this singular malady—the breakbone fever of parts of our country—is unknown. McLaughlin[1] has found in the blood micrococci in great numbers, about one-twentieth to one-thirtieth the diameter of the red corpuscles, of spherical shape and red or purplish in color.

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[1] Journ. Amer. Med. Assoc., June 19, 1886.

Dengue is generally a harmless disorder, epidemic, and contagious. Isolated cases are difficult of diagnosis, but when the disease largely prevails its recognition is easy. It differs from *rheumatism* or *gout* by the significant features of the fever and the eruption; from *scarlet fever* by the different character and want of continuity of the fever, by the pains, the arthritic symptoms, and the polymorphous eruption towards the close; from *influenza* by these, and chiefly by the eruption. The remission may cause the disease to be mistaken for a *malarial fever;* but the irregularity of the fever in dengue, the joint and muscle pains, the rashes, and the absence of hepatic and splenic enlargement are very unlike. Dengue has a closer resemblance to *yellow fever*, and the difficulty of distinction becomes the greater because epidemics of both may be present side by side, and because we may find most of the same symptoms, even the jaundice, the albuminous urine, the hemorrhages, and the slow pulse with elevated temperature. But all these signs are of comparatively infrequent occurrence, and neither jaundice nor albuminuria is an early symptom, as in yellow fever. Moreover, the single paroxysm, the tongue with red edges, the great irritability of the stomach, the grave nervous symptoms are not met with in dengue; and, on the other hand, we miss in yellow fever the rashes, and the pains and swelling of the joints. Dengue is not a serious disease; yellow fever is a very dangerous one, and the character of the prevailing epidemic is mostly conclusive. But when they coexist, the distinction between a light case of the latter and a severe case of the former may be very difficult.

Plague.—The plague, also known as bubonic plague or the pest, is an acute infective fever accompanied by inflammatory swelling of the lymphatic glands, and is due to a micro-organism, the *bacillus pestis*, discovered by Kitasato, of Japan. It is a disease that prevailed in frightful epidemics in the Middle Ages, in Europe as well as the East, and was popularly called the "black death." Now it is unknown in Europe and this country, except for a few sporadic cases that have been imported or have been developed in bacteriological laboratories, and the epidemic at Astrachan, in Russia; though quite recently there have been some cases in Portugal. In parts of Asia, especially of India and China, it is still prevalent.

There are two forms in which plague shows itself,—the severe or ordinary plague, *pestis major*, and a minor or abortive form. The ordinary plague is a highly contagious malady, and spreads as an epidemic. It has a short period of incubation, not more than one week. Its early symptoms are headache, vertigo, and staggering gait;

the face is pallid and vacant; the eye is injected; the patient appears stupefied by the poison. There is from the onset extreme muscular weakness; soon high fever shows itself, preceded by chilly sensations or a chill. The temperature is high, and may range between 104° and 107°; in favorable cases it falls gradually. There is great thirst, as well as burning in the throat and stomach. The pulse is rapid, generally weak; the bowels are constipated. There is stupor, or coma. The febrile stage does not generally exceed five days. Before its conclusion, sometimes from the start, buboes appear, often attended with some abatement of the general symptoms. The glands are hard and painful, and frequently surrounded by œdematous skin; their slow suppuration is looked upon as favorable. Not only the inguinal glands, but the femoral, the axillary, the submaxillary, and other lymph-glands may be attacked. The glandular affection outlasts the febrile stage. Purpuric spots and petechiæ, and hemorrhages from various parts, especially from the lungs and bowels, are also at times noticed, as is bilious vomiting.

The disease, mostly fatal, is a short one, generally lasting from three to five days, though suppuration in the buboes may keep ill for a long time even the cases that recover from the fever. The short duration of the febrile malady, the absence of a characteristic eruption, the presence of buboes, distinguish it from typhus fever. From forms of malignant malarial fever, for which it has been sometimes mistaken, it differs by the signs of the affection of the lymph-glands, the absence of intermissions or decided remissions and of malarial organisms in the blood; on the other hand, the bacillus of plague can be detected in the lymph-glands.

The minor form, *pestis minor*, has but slight fever, and no violent symptoms. The glandular swellings are its only marked sign. It is rather endemic than epidemic, though it sometimes has been noticed to precede ordinary epidemic plague, which, it is thought, may develop from it. The minor form of plague lasts about two weeks; it is very rarely fatal, and is supposed not to be contagious.

Malta Fever.—This is a disease known also as the Mediterranean fever, "rock fever," Neapolitan fever, and by many other names. There is reason to believe that it also exists in Porto Rico, and its occurrence has been recently established in the United States.[1] The disease is an infectious fever of hot climates, due to a micro-organism

[1] Case of Musser and Sailer, Phila. Med. Journal, Dec. 31, 1898. The case reported by A. A. Smith, Trans. Assoc. Amer. Phys., 1897, as Levant fever, and in which a non-malarial parasite was found in the blood, is also, most probably, an illustration of the disease.

described by Bruce, the *micrococcus Melitensis*, and is found in association with bad sewerage. It is generally met with in epidemics, in which the mortality is not great, and which alternate with typhoid fever. It mostly begins gradually, with languor, chilliness, weakness, and muscular pains, but rarely with a chill or vomiting. Symptoms of gastric and intestinal catarrh appear early and continue throughout. There is enlargement of the spleen with tenderness, also muscular pain and marked anæmia. The tonsils are often swollen; the bowels are generally constipated. Palpitation is of common occurrence, and hæmic murmurs are heard. Epistaxis, bleeding from the gums, and hæmoptysis are usual. Bruce has proved that while the red corpuscles diminish greatly, the white corpuscles, as in typhoid fever, remain in normal amount. The temperature is that of a continued fever, generally between 102° and 104°, but very irregular. There is profuse perspiration, also great restlessness, weariness, and often insomnia; orchitis is not uncommon. After a week, or longer, the symptoms decline, and the patient appears to be convalescing, but in a few days a relapse is apt to happen with recurrence of the marked symptoms. In this relapse the fever may assume a remittent rather than a continuous form. These relapses may be frequently repeated, and thus the disease be a very protracted one. Late in the original attack or in the relapse there are rheumatic pains in the joints, especially, as found by Notter,[1] in the ankle and sacro-ileal joints, which become very tender, and at times the seat of an effusion. Node-like swellings occur on the ribs and on the costal cartilages. It will be seen from this description that the disease simulates dengue, but the peculiar eruptions of this are absent, and the arthritic symptoms occur later, nor is jaundice present. Moreover, the finding of the micrococcus Melitensis is conclusive. There is a serum test producing agglutination, similar to that obtained in typhoid fever.[2]

In rare and very protracted cases the swelling of the joints may lead to the supposition of a typhoid fever with arthritic complications and with relapses. In such a case,[3] in which Malta fever was suspected, that occurred in my ward at the Pennsylvania Hospital last winter, the blood examination made by Doctors Kirkbride and Kneass proved it to be not Malta fever, but typhoid fever with arthritic complications. There was a positive reaction with the Widal test, but no characteristic signs of Malta fever with the special serum test for this.

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[1] Allbutt's System of Medicine, vol. ii., article "Malta Fever."

[2] Wright, The Lancet, March, 1897.

[3] Philadelphia Medical Journal, May 6, 1899.

Glandular Fever.—This disease, first described by Pfeiffer, is an infectious fever in children associated with marked swelling of the lymphatic glands, especially those of the neck. The fever is generally pronounced, 101° to 103°, but of short duration; the swelling of the glands persists for several weeks. Not only the cervical glands are swollen, but frequently also the axillary and the inguinal glands. Both spleen and liver are mostly enlarged; there is slight redness of the throat. The fever precedes the tenderness and swelling of the glands by a day or two; at times there is puffiness of the skin around them, and they may suppurate. Nephritis is an occasional complication. The disease nearly always ends favorably.

Periodical Fevers.

These fevers are characterized by the distinct periodicity of their phenomena: they exhibit intervals during which the patient is wholly or nearly free from febrile disturbance; they are all owing to malaria. This noxious agent gives rise to a group of fevers ever betraying their common origin by their strong family resemblance: alike in occurring in low, swampy localities; alike in most of their symptoms, and in the difficulty of eradication from the system; alike in being due to well-recognized micro-organisms; alike in the secondary lesions, in the enlargement of the spleen and of the liver, and in the altered condition of the blood, which they leave behind them; and also alike in being under the control, absolute and immediate, of cinchona and its various preparations.

Since the great discovery by Laveran of the malarial parasite our knowledge of all malarial fevers has become much clearer, and infinitely more exact, and this shows itself as much in diagnosis as in pathological studies. It is, therefore, fitting that a short description of the malarial organisms should precede the description of the individual fevers, at least in so far as they concern questions of diagnosis; for the larger questions of origin, growth,[1] and technical study I must refer to the admirable works of Thayer and of Mannaberg,[2] and to the numerous papers of observers, such as Marchiafava, Celli, Golgi, Grassi, Sternberg, Dock, Hewetson, and Manson, who have done so much to extend our knowledge.

The malarial parasite is best studied in fresh blood, care being taken that the cover-glasses and slides have been well cleansed in alcohol or ether; a drop of blood is readily obtained from the lobe of

[1] Malarial Fevers, 1897.

[2] In Nothnagel's Spec. Path. u. Therap., 1899.

the ear, thoroughly washed. If stains be employed, Loeffler's methylene-blue is very serviceable. There are three forms of parasites now recognized, which pass through their cycle of development in from twenty-four to seventy-two hours; at the start they are small, colorless bodies within the red corpuscles and are soon seen to be actively amœboid. As the bodies increase in size, pigment granules dot their periphery; gradually the centre or very nearly the entire red corpuscle is taken up; the pigment in the organism increases, and becomes darker and coarser. After full development has been reached, sporulation takes place, and the pigment mostly collects into a small mass at a particular point, generally towards the centre. The red corpuscle now bursts and the segments or spores are set free, and invade fresh corpuscles; the pigment granules float in the blood-serum. But the parasite may escape from the red blood-cell before sporulation. In some instances roundish vacuoles of irregular size, thought to be due to degenerative process, are observed in the parasite; or thread-like, colorless, actively motile flagella appear from the periphery of the organism.

Different forms of malarial parasites produce different types of malarial fever, and there is a close connection between their development and the clinical features of the fever; the paroxysms, as pointed out by Golgi, are associated with the segmentation of a group of the malarial parasites. Very often there is evidence of two or more groups, and if these reach maturity on different days and at different times types of fever are produced entirely different from those when the groups are single. A combination of the main malarial organisms may also occur.

There are three distinct varieties of malarial parasites, and some subdivisions; the three distinct and chief varieties are:

1. The parasite of tertian fever.
2. The parasite of quartan fever.
3. The parasite of æstivo-autumnal fever.

1. The *tertian* parasite, by far the most frequently observed in this country, completes its cycle of development in forty-eight hours. It is larger, less refractive, has much more active amœboid motion than the quartan, and has finer, lighter pigment granules and rods; the pigment moves very markedly. The red corpuscle containing the parasite swells up, and becomes paler than normal. The pigment at maturity is collected into a mass near the centre, and the parasite is absolutely quiescent; it breaks up into fifteen to twenty segments, and the spores are rounded and smaller than those of the quartan parasite.

DESCRIPTION OF PLATE VI.

MALARIAL PARASITES.

A number of these micro-organisms were obtained in blood examinations of malarial fevers made at the Pennsylvania Hospital, and drawn from nature by Dr. C. F. M. Leidy. Some of the rarer forms, especially of the æstivo-autumnal variety, are taken from the works of Mannaberg and of Thayer. The engraving is by Mr. Louis Schmidt.

In the *tertian* group, the first is a red corpuscle of normal size. The swelling of the corpuscle by the tertian parasite is seen in the following ones.

The second and third show hyaline bodies; the next four, the gradual growth and development of the parasite and pigmentation in the same; then follow segmentation and discharge of the spores, of which there are from fifteen to twenty. The last body is a large flagellate.

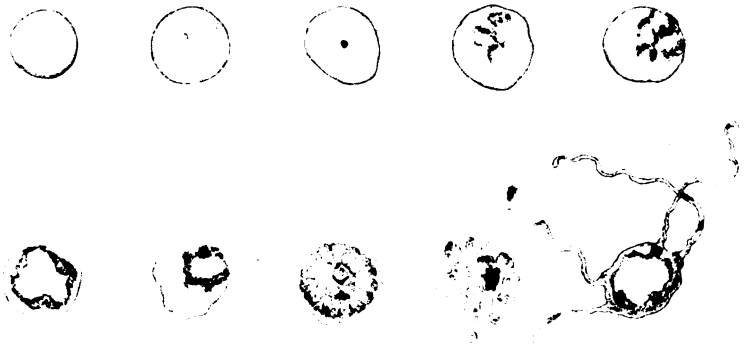
In the *quartan* group are shown different forms of the quartan parasite, their development and segmentation. The parasite is small and the corpuscle has a tendency to contract around it, the rim having a deep coloration. The pigment is coarser and darker than in the tertian, and there are only from six to twelve sporules in segmentation. The flagellate is smaller than the tertian.

In the *æstivo-autumnal* form the pigmentation is seen to be more marked towards the periphery of the parasite. The figures show the small size of this parasite, which is the smallest of the malarial parasites, but always very distinct. In the fourth of this group the degeneration of the corpuscle is distinctly perceived. On the last line various forms of ovals and crescents are seen as well as a flagellate. The flagellate is coarsely pigmented, but smaller than the tertian variety.

PLATE VI.

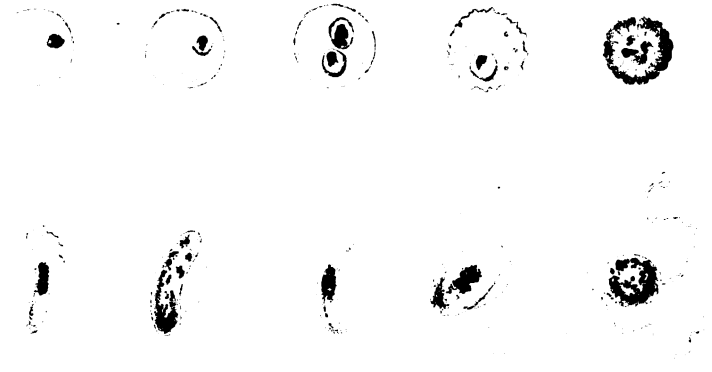
MALARIAL PARASITES.

TERTIAN FORMS.



QUARTAN FORMS.

ÆSTIVO-AUTUMNAL FORMS.



2. The parasite of *quartan* fever has a cycle of development of about seventy-two hours; sporulation occurs every fourth day. The pigment is coarse and dark and found on one side chiefly; the parasite and the pigment have slow motions. The young parasite is small, about one-fourth the size of a red blood-corpuscle. As the parasite grows, the red corpuscle contracts around it, and the rim shows deep coloration; there is no irregular breaking up of the organism into sections, of which there are from six to twelve. Before segmentation, the pigment tends to the centre in radial lines, forming a star-like arrangement. When two groups of organisms reach maturity on different days we have paroxysms on successive days and a day of intermission.

3. The *æstivo-autumnal* parasite is the most irregular of all the malarial parasites; the cycle of development varies from twenty-four to forty-eight hours, and it does not, like the other forms, occur in great groups which arrive at maturity at the same time. Its most distinctive feature is the production of crescents from the spherical parasite within the red corpuscle. These crescents are very generally pigmented; but the bodies may be oval or fusiform in place of crescentic. The crescents are not met with unless the fever be at least of a week's duration. In their earliest stages the æstivo-autumnal organisms are like the tertian or quartan forms, except smaller, and they often first show themselves as minute ring-like refractive bodies, in which a few dark-brown pigment granules appear, and the red corpuscles soon exhibit degenerative changes. The pigment gathers towards the centre, and segmentation takes place as in the tertian parasite. But segmentation is very rarely seen in the blood taken from the peripheral circulation, indeed, only the youngest form of the parasite and the crescents are encountered; the later development of the organism and the segmented bodies can be studied in blood taken from the spleen. The irregularity of development and maturity accounts for the irregularity of the clinical manifestations in the malarial fevers in which the æstivo-autumnal parasite is found. This is, indeed, pre-eminently the malarial organism of all irregular exhibitions of malarial infection, as well as of the autumnal malarial remittent fever which is so varied in form. The parasite has been further divided into two varieties, the quotidian and malignant tertian organisms, and these have been further subdivided as to whether pigmented or not. But these distinctions are not generally accepted, and their clinical value has not been determined.

We shall now look at the clinical side of malarial fevers, premising that it is the general tendency of malarial paroxysms to anticipate.

Intermittent Fever.—The paroxysm comes on with a chill: the face becomes pale, the lips bluish; the teeth chatter; the skin is cold; there is a feeling of uneasiness and fatigue. After a period varying commonly from half an hour to an hour, this cold stage passes off. Now we find decided heat of the surface, with restlessness, thirst, a full, rapid pulse, muscular pains, a scanty secretion of urine; in other words, active febrile symptoms. These continue for hours, for a period always much longer than the first stage: then a sweat breaks out all over the body; the pulse becomes softer and less frequent; the secretions are fully re-established; and this sweating stage terminates the paroxysm.

The patient is now, for the time being, well; but the disease soon recurs: in from twenty-four to seventy-two hours the paroxysm repeats itself. In the former case we call the fever a *quotidian;* in the latter, a *quartan*. The *tertian* type is before us when the paroxysm sets in again in about forty-eight hours; the *double tertian*, when we find a daily attack, but those of alternate days alone corresponding in time and severity. Even a *quintan* ague may happen.[1] The period between the ending of one attack and the beginning of another is spoken of as the *intermission* or *apyrexia;* while the time between the beginning of the two paroxysms, including the first with its succeeding intermission, is called the *interval.*

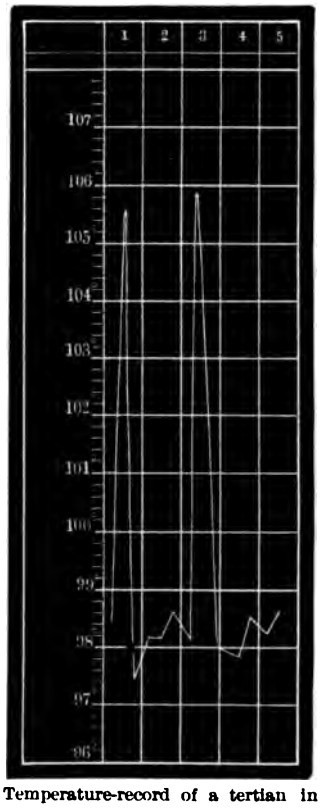
The tertian and the quotidian are the usual types in this country. In the ordinary tertian there is a single group of infection with the tertian malarial organism; where the quartan parasite is present in large numbers and as a single infection that reaches maturity at about the same time, we have the quartan fever; if in two groups, reaching their full development on successive days, with a day of intermission, the double quartan. Should either the tertian or the quartan parasite occur, the first in double, the second in triple infection,—the different sets of parasites reaching maturity on successive days,—we have the quotidian type of the fever, which may thus depend on either the tertian or quartan parasite; or, again, there may be a coexistence of these, which is not, however, frequent. Even the æstivo-autumnal infection may produce quotidian intermittents. Yet this is not common, and the paroxysms are much less regular. The most usual cause of the quotidian in this country is the infection with two groups of tertian parasites that reach maturity on successive days.

The varied types of the fever present marked differences in the character and duration of the several stages. The tertian has gener-

[1] Case of Henry, Brit. Med. Journ., Feb. 18, 1888.

ally the longest hot stage, the quartan the longest cold stage. In the quotidian there is a short cold stage, followed by a hot stage which may last for upward of fifteen hours. Occasionally the stages are very irregular and anomalous. Thus, the sweating stage may precede the cold stage, or it may be the only one which shows itself; or, again, the rigor may be altogether wanting. Sometimes, there are no distinct stages, but the patient has a "dumb ague," which manifests itself at definite periods by a feeling of great depression, or of a severe pain at some portion of the body, or by chilly sensations, or by headache, or by nausea and vomiting, or, as I have seen, by excruciating pain over the kidneys, and almost entire suppression of urine, or by spasmodic obstruction of the intestine.[1]

FIG. 80.



Temperature-record of a tertian intermittent.

The temperature in intermittent fever shows a record that, in doubtful cases, may be turned to great advantage. Notwithstanding the marked sense of chilliness, the thermometer rises suddenly and rapidly to a high degree; there may be a slight elevation of temperature for an hour before a chill, but the striking rise begins with the chill. Even during the decided chill of the beginning of the paroxysm it indicates 105° or more in the axilla. The temperature remains stationary, or continues to rise, though not much, during the hot stage, and during the sweating stage falls at first slowly, then rapidly, until it comes down to about the normal heat. During the chill the peripheral temperature is decidedly lowered; during the hot stage it is increased. But with the ending of the paroxysm it is found that the fall has been rapid. In the intermission the thermometer in the axilla marks a natural temperature, or one somewhat lower than in health. It rises again quickly with each paroxysm. No other malady presents these variations.

In some cases of intermittent fever an intermitting murmur is

[1] Cases of Hoyt, Atlanta Med. and Surg. Journ., Sept. 1875.

heard over the spleen. This is ascribed to the movement of the blood in the splenic arteries with the systole, in consequence of the soft, enlarged, and overfilled condition of the spleen. It is usually detected most distinctly during the febrile period, ceases with the paroxysms,[1] and is not heard in chronic malaria.

To the peculiar appearance of the tongue which those under the malarial influence may show, Osborn has directed particular attention.[2] There is a distinct lateral boundary of the organ, an appearance of indentation transversely, and the inferior surface appears to have encroached upon the superior and lateral borders.

The diagnosis of an ordinary and regular intermittent is easy. Leaving the other malarial fevers out of consideration, only two morbid states are likely to present recurring rigors and febrile excitement, and are, therefore, apt to be confounded with it: hectic fever, and chills attending upon suppuration in deep-seated parts. Now, *hectic fever* differs in this from intermittent: it is simply a fever of irritation, the cause of which a careful scrutiny will generally detect. We find it accompanying many chronic diseases in which destruction of tissue occurs, especially phthisis; and the chronic affection has its own signs, which exist at all times, whether the symptomatic fever be present or not. Then its outbreaks are irregular. Several often take place within the twenty-four hours; their intermissions are incomplete; the temperature does not fall as in intermittent fever, for there is not complete defervescence; and although the paroxysms may begin with chilliness, they are not ushered in by a well-defined rigor. Further, they are apt to be morning paroxysms, and are not modified by antiperiodics. Whenever, indeed, we find an intermitting fever not influenced by these agents, it ought to arouse suspicion, and all the internal organs, particularly the lungs, should be carefully explored. Thus only can serious errors in diagnosis be guarded against.

When *pus* forms, and especially when it forms in internal cavities, it betrays its presence by rigors, followed by more or less fever. But these, unlike the chills of ague, do not repeat themselves at definite periods. Moreover, in the midst of the apparent intermission, febrile signs or other manifestations of a seriously disordered system may be discovered; or we may find the local cause, for instance, a pelvic cellulitis. The chills of ordinary pyæmia, unlike the malarial malady, are often characterized by the profuse sweating that immediately follows them, rather than by an active development of fever. In cases of

[1] Maissurianz, St. Petersburger Medicin. Wochenschr., 1882, 12.

[2] Transactions of the American Medical Association, vol. xx.

purulent collections and infection we have marked leucocytosis, but of even greater value in diagnosis is the absence of the malarial organisms.

There are other causes which may occasion attacks of fever happening in paroxysms and simulating ague, though not malarial. They may occur in *diseases of the heart*, as in ulcerative endocarditis and in valvular affections.[1] *Gall-stones* which form in the radicals of the hepatic duct in the interior of the liver may, as Frerichs shows, give rise to attacks of chills, followed by heat and by sweating, easily mistaken for ague. The fact that these febrile phenomena are preceded in many cases of *intrahepatic concretion* by dull pain in the hepatic region, and by sudden sharp seizures of pain at the lower part of the thorax on the right side, is very significant. Then we have the ordinary form of *hepatic fever*, which we have already discussed, and of which impacted gall-stone is the most common cause, and recurring jaundice, with more or less pain, the main symptom. The paroxysms often come at first with some regularity, and are more likely to be repeated in the afternoon and evening, while the malarial paroxysm more commonly occurs in the morning.

An affection which on account of the chill succeeded by fever might be mistaken for the malarial disorder is the curious so-called *urethral fever* which sometimes arises after the passage of a bougie, and which may even terminate in death.[2] Our knowledge of the introduction of the instrument, and the non-recurrence at a fixed time of the rigor and febrile phenomena, furnish the points of distinction.

Yet another affection liable to be mistaken for intermittent fever is *syphilitic fever*. The fever may occur in attacks consisting of a chill, followed by a hot stage and sweating, and be so similar to the malarial disorder as to lead to error.[3] The apparent ague-fits happen, however, towards evening, and are succeeded or accompanied by severe headache and pains in the bones,—in fact, by the same symptoms as the more ordinary kind of syphilitic fever. In the form in which the febrile symptoms are continuous, these generally precede the eruption for a week or more, and may continue after this appears; but an eruption may be found, and the history of syphilis be doubtful. In these obscure cases the fever lasts a long time, the pulse-rate

[1] Osler, Practitioner, vol. i., No. 3, p. 181; Henry, Amer. Journ. Med. Sci., July, 1899, has reported a case of mitral stenosis with fever recurring at intervals of about a week.

[2] Roser, quoted in Brit. and For. Med.-Chir. Rev., Oct. 1867.

[3] See cases of Bassereau, referred to by Bumstead in his Treatise on Venereal Diseases; Ord, *loc. cit.*

is slow, and they gradually yield to antisyphilitic treatment, while by repeated examinations of the blood anæmia, but no malarial parasites, are detected.[1]

We may also find syphilitic fever in cerebral syphilis with symptoms like those of malaria.[2] The paroxysmal pyrexias may be met with at very varying times after the infection, though, like everything connected with cerebral syphilis, they are generally late manifestations. There is often a preceding history of severe headache, of irregular motor palsies and epileptic attacks, of mental failure and perversion, or of symptoms similar to general paralysis, though wanting in the tremulousness. The aphasia which may be met with is said to be commonly associated with left-sided hemiplegia.

Syphilitic fever is, on the whole, less apt to be confounded with malarial disease than it is with tubercular affections; a very common error, as Janeway has proved.[3] Long-continued, causeless fever in which blood-examinations show no malarial organisms, and where there is no distinct evidence of tuberculosis, should always make us very suspicious of syphilis.

In the diagnosis of intermittent fever we have also to consider that certain diseases which are non-malarial exhibit at times a deceptive *periodicity;* they may be worse every second day. Even mania, as Schroeder van der Kolk has pointed out, may take this type. In all such instances the microscopic examination of the blood for malarial parasites is of the greatest value.

In the puerperal state a malarial outbreak may happen which, as Manson and Fordyce Barker[4] have shown, may be mistaken for puerperal fever. Unlike the latter, however, the *puerperal malarial* fever is attended with pain in the head, back, and limbs, and does not generally appear so soon after parturition,—not, therefore, between the first and fifth days after delivery. Moreover, it has at the beginning a great temperature-rise, and marked remissions or intermissions. Puerperal malarial fever may lead, after the twelfth day, to secondary hemorrhage.

Now, in all these diseases simulating outbreaks there are two tests of great value, more important than any mentioned,—one the therapeutic test of their not yielding to decided doses of quinine; the other, still more valuable, that careful and repeated examinations of the

[1] Cases of Musser and of Prentiss, Phila. Med. Journ., July, 1899.

[2] Wood, Transactions of the College of Physicians of Philadelphia, Feb. 1884; also in Medical News, Philadelphia, March, 1881; Janowsky, quoted *ibid.*

[3] Transactions of the Association of American Physicians, 1898.

[4] Medical Record, Feb. 1880; Virginia Med. Monthly, Nov. 1881.

blood fail to detect the malarial organisms. Further, though not so generally applicable in complicated malarial fevers, there is no leucocytosis, while decided leucocytosis is among the features of most of the conditions named, and especially of all those with a septic infection.

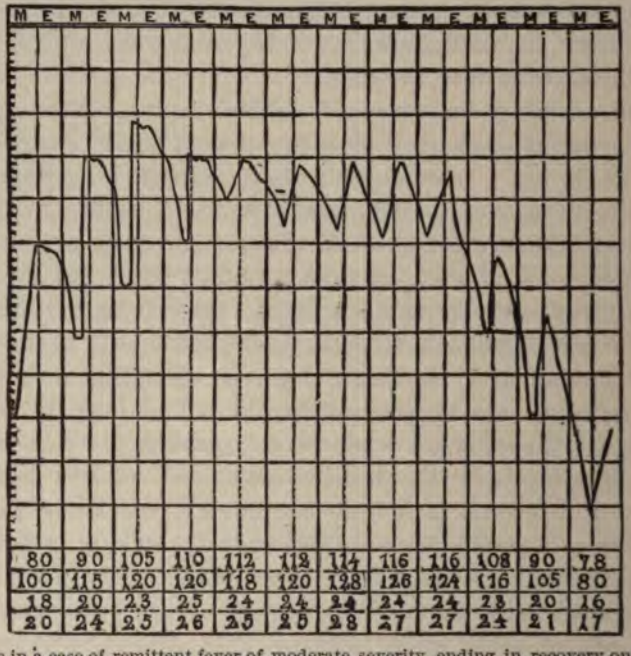
Remittent Fever.—This is a fever pre-eminently of hot climates and malarial districts, and is now more generally described as æstivo-autumnal fever. It is the fever of Hungary, of the Pontine Marshes, and particularly of Africa and the southern portion of the North American continent, and of parts of South America. Occasionally, not often, we meet with it in winter and in early spring; very generally, during the summer and autumn months. The malarial parasite that occasions it is the æstivo-autumnal parasite, of which, as above stated, a main characteristic is irregularity, and we see this reproduced in the clinical features of the disease.

Remittent fever has no well-defined and constant prodromic symptoms, except, perhaps, a singular sense of gastric uneasiness. It is ushered in by a marked chill, soon succeeded by violent fever, which, after a varying period, decreases, and then breaks out again. By this time the symptoms of the disease are very apparent. The patient complains of pain, of fulness and of throbbing in his head. He is restless and distressed; his limbs ache; his tongue has become coated; he suffers from thirst, and rejects the contents of the stomach. After continuing at their height from six to eighteen hours, these symptoms again subside: a sweat breaks out all over the body; the irritability of the stomach lessens; the patient is composed, even cheerful; his headache has nearly ceased, and he falls into a quiet slumber. But this lull is not of long duration, not longer than some hours. Soon the active fever is rekindled: the skin is as hot and dry as before, the pulse as full, frequent, and hard; the spleen is observed to be swollen; and the other symptoms return with increased intensity, again to abate, again to recur, until either the exacerbations are effaced and the fever assumes a continued type, or else the remissions become better and better defined,—more, indeed, like intermissions than remissions. In the progress of the disease and after its height the pulse is generally quicker and weaker than at first.

The temperature rises markedly with the first chill, and continues to rise during the high fever that follows. With the sweating stage it declines by several degrees, to rise to a greater height than previously with the succeeding febrile phenomena; then again there is a fall in the remission, with another quick rise in the fever, which may attain a very high point, marking from 105° to 108°. The greatest height

is usually reached in the exacerbation of the third day. After this the remissions become less distinct, and may be, indeed, recognizable only by the thermometer; the whole fever is more like a continued fever. Subsequently to the ninth day usually the remissions are very marked, the difference between the temperature in them and the exacerbations being three degrees or more. The exacerbations become less and less high, and soon cease, the temperature falling perhaps previously to below the norm. In cases in which the fever remains for a long time continuous, irregular remissions occur, especially towards the end, though the fever may preserve its continued type until it gradually ceases.

FIG. 81.



Temperature in a case of remittent fever of moderate severity, ending in recovery on the twelfth day. The chart shows also the pulse and the respiration.

The average duration of the fever, unless protracted by complications, is from nine to twelve days. Its most common form is quotidian, or rather, perhaps, double tertian, the exacerbations of alternate days corresponding in severity, in duration, and even in the nature of the symptoms. Sometimes there are two exacerbations in twenty-four hours,—a duplicated quotidian,—or the paroxysms have a tertian form. The exacerbations may occur any time in the twenty-four hours; in many instances morning exacerbation is noticed, and I have

met with more cases in which the paroxysm comes on in the afternoon than in the evening.

The urine in remittent fever presents much the same changes, though in a different degree, as those occurring in intermittent fever. During the active stages of the fever there is an increase of urea, not simply above the standard of health, but even above that in intermittent fever; and this increase of urea is attended with a diminution of uric acid—unlike what happens during the paroxysms of ague—and of the coloring and extractive matter; while, as convalescence sets in, the urea decreases in amount, and the other ingredients mentioned increase.[1] A copious deposit of urates, forming with the phosphates as it were a critical discharge, is noticed as the fever subsides, and is analogous to what takes place after the paroxysm in intermittent fever. At no stage does the urine contain albumin, as it often does in typhus, and as it generally does in yellow fever; but, as in intermittent, it may contain sugar.

Remittent fever is readily recognized: the rise and fall of its febrile signs are too striking to escape observation. Its characteristic traits are closely allied to those of *intermittent fever*. But there are these points of contrast: in intermittent fever each paroxysm begins with a chill, which is not the case in remittent fever; for after the first paroxysm there is rarely a marked chill, and even the chill ushering in the disease is usually not violent. After each febrile exacerbation comes an abatement,—not an intermission, for the thermometer shows that the fever does not leave; the tongue remains coated, and the gastric derangement does not entirely cease; the patient is not well. The symptoms rise and decline; they do not, as in ague, appear and disappear. In both affections we may have herpes labialis at the decline, but it is more common in remittent than in intermittent.

Owing to the jaundice in many cases of bilious remittent fever, the disease is often mistaken for *acute congestion of the liver*, or *acute catarrhal jaundice*. Here, again, the exacerbations and remissions in the temperature serve as distinguishing marks; and so, too, in separating the gastric complications of bilious remittent fever from acute gastric inflammation. The severe headache is also a distinctive feature of value; so is the herpes labialis. But of greatest importance and conclusive is the finding of the malarial parasites.

Under ordinary circumstances there is very little likelihood of confounding with each other *typhoid* and remittent fevers. The lines between the two diseases are too strongly drawn: no marked perio-

[1] Joseph Jones, Observations on Malarial Fever.

dicity exists in typhoid fever, nor are vomiting and jaundice often seen; and, on the other hand, we find no diarrhœa, no eruption, except at times herpes and urticaria, no thoracic symptoms, no deafness, and no very great prostration in remittent fever, and the symptoms are strikingly influenced by quinine. Very decided periodicity may be witnessed in typhoid fever as it is approaching a favorable termination; the afternoon or evening rise of temperature is most marked, the morning remission very great. Here a knowledge of the previous history of the case and the Widal test guard against error. We shall presently again refer to the symptoms of periodicity in examining into typho-malarial fever.

Further, not infrequently, after an attack of remittent fever has lasted for ten or twelve days, these symptoms are noticed: great muscular debility, jerking of the tendons, picking at the bedclothes, dark, dry tongue, and weak pulse, perhaps diarrhœa. The fever becomes of a continued type. It is these cases which have given rise to the opinion that bilious fever often changes into typhoid fever. But in reality it is not the specific typhoid fever, with its enteric lesions, but a typhoid condition, that is developed. The Widal test is negative; malarial organisms are found in the blood.

During the exacerbations of remittent fever the cerebral symptoms are sometimes almost identical with those of an acute brain-affection. There is severe headache, with violent beating of the arteries of the neck and face, a wild eye, intolerance of light, and even delirium. Were the patient now seen for the first time, he would be pronounced to be laboring under *acute meningitis*. Suddenly the pulse loses its throbbing character, a perspiration covers the surface, and, unexpectedly, the cerebral disturbance ceases until the next paroxysm redevelops it. Cases of this kind are readily enough recognized, if we know something of their history. If we are not familiar with it, we have to await the remission for their explanation; and after the sudden cessation of the signs of disorder of the brain it is hardly possible to have doubts as to the meaning of the acute nervous symptoms, should they recur. But occasionally these show themselves under circumstances where a malarial poison is not suspected to be at work:

A young gentleman of studious habits, while diligently preparing for a college examination, was seized with violent headache and fever. The sense of fulness in the head was unbearable, the fever was high, there was nausea with great gastric irritability. These symptoms lasted for nearly twenty-four hours, and then subsided in the forenoon, to become aggravated in the evening. Delirium followed by great drowsiness was perceived at an early hour of the third day

of the disease. The case now assumed a very alarming aspect. Local bloodletting was resorted to with some relief, and in a few hours the symptoms were, fortunately, favorably modified: the headache was much less, the mind was again quite clear. Although the patient had never suffered from a malarial fever, he had spent part of his summer vacation in the marshy neighborhood of Washington; but several months had elapsed, and winter was setting in. The time of the year was not in favor of malaria. But the evident remission in the cerebral symptoms, the coated state of the tongue, and the malarial look of the countenance, that became daily more apparent, decided me upon administering quinine. The evening exacerbation came, but was far less severe. The nature of the case was now evident: the quinine treatment was vigorously pursued, and the patient soon recovered.

The violent headache and delirium were in this case observed to be in connection with well-defined febrile signs. Occasionally one or both of the symptoms mentioned last during remission, while the fever abates. I have even met with them occurring in paroxysms without fever being present, as in the following case seen a number of years ago:

A young lady of delicate constitution was attacked, in September, with remittent fever. The disease ran its course without any unusual symptoms; a violent headache, but little, if any, wandering of the mind being observed during the daily exacerbations. After the tenth day the fever lessened, and the disease assumed a continued type; yet soon afterwards, as convalescence seemed to be established, every evening for three days, between five and six o'clock, a boisterous delirium set in, lasting for three or four hours, and once nearly all night. It was followed by a profound sleep, from which she woke up with a clear mind. During these fits the pulse was not accelerated, and there was no fever. The third attack was not so very severe, as the patient was already in part under the influence of decided doses of quinine; another was prevented by this drug.

Both these cases were seen before the discovery of the malarial parasite; the presence of this would have at once determined their true nature. In both the symptoms approached those of the congestive type of the disease, and the issue appeared at one time doubtful. Generally speaking, remittent fever, unless it be of the congestive variety, has a favorable prognosis. It is difficult for us, living in a century in which the remarkable effects of quinine are so well understood, to believe that the complaint was once so fatal, and that so many deaths should have taken place from a disorder over which we now exercise so undoubted a control. But the long list of dis-

tinguished names that have fallen victims to it, among them Cromwell, James I., and the Emperor Charles V.,[1] proves the medical skill of former times to have been insufficient for its cure. In our day, the consequences of remittent fever are more to be dreaded than the disease itself. We often find, as its sequelæ, obstinate intermittents, enlargement of the liver and spleen, dropsy, protracted anæmia, headache, and impaired activity of mind.

In children, a fever of remittent type is observed, called *infantile remittent*, which is rarely a miasmatic disorder. It is often a gastro-enteritis connected with verminous irritation or produced by errors in diet; or a typhoid fever,—an affection which now and then occurs even in very young children. What has given rise to this confusion is, that all febrile diseases in children exhibit a much greater periodicity than in adults, and in all some cerebral symptoms are apt to be present. To distinguish the two maladies mentioned from true remittent fever, we must study particularly their manner of beginning and their probable origin, and note the peculiarities of the abdominal symptoms. Then we may lay stress on the irregular mode and the unequal duration of the febrile exacerbations. Sometimes, also, by close scrutiny, the characteristic eruption of a low continued fever may be found in an apparent remittent.

But some of these cases of infantile remittent fever are really of malarial origin; even in young children this may be their source. I saw, for instance, some years ago, a little girl, three years of age, who had a distinctly malarial remittent fever, which was checked by antiperiodics. During the violent exacerbations she was very delirious; her face had a most anxious, frightened look; her screams could be heard all over the house. In the remissions she was perfectly sensible, but there was gastric irritability, and the bowels were very constipated. I have met with a similar case in an infant of eighteen months.

Pernicious or Congestive Fever.—This is a malignant, malarial fever, which may be either of the intermittent or of the remittent form, and with rare exceptions depends upon infection with the æstivo-autumnal parasite which is present in large numbers. A special form of the æstivo-autumnal parasite, the malignant tertian parasite, is held to be the cause of the malignancy. But this is not certain. Mannaberg lays stress on individual predisposition and on the anatomical

[1] From the record of the Emperor's illness, as given by the historian Mignet (Charles V au Monastère de Yuste), we may learn, what fortunately now we hardly have an opportunity of observing, the features of remittent fever when left to itself.

lesions produced, such as occlusion of the finer blood-vessels with the infected blood-corpuscles. If ordinary æstivo-autumnal fever be not treated, it tends to become pernicious. The pernicious attacks are of the tertian or the quotidian type. While they are at their height, there is intense congestion of one or several internal organs, with a dangerous perversion of the function of innervation. From this state the patient may rally, but only to fall a victim to another paroxysm, unless art intervene. The temperature during the chill and subsequent fever ranges from 104° to 108°. Sugar is found in the urine much more commonly than in ordinary intermittent fever.

The symptoms of this violent malady vary according to the organ more specially disturbed, and to the extent of the derangement of the nervous system. We have, thus, several distinct varieties, of which I shall describe the prominent.

The *gastro-enteric* form is common in our Southwestern States. Its distinctive features are nausea and vomiting, purging of thin discharges mixed with blood, intense thirst, and an equally intense desire for air. There is little abdominal pain or tenderness, but a weak, frequent pulse, and very great restlessness. The patient complains of a sense of sinking and of weight, and of burning heat in the stomach. His breathing is deep-drawn; to each expiration succeed two short inspirations. The face, hands, and feet are pale and cold; the features shrunken. Sometimes these symptoms continue for several days, and gradually increase in intensity, in spite of nature making efforts at reaction. More frequently reaction does take place; the temperature is very high, the pulse feeble, and the stormy symptoms subside or wholly yield, until another outbreak, which is very apt to be deadly, occurs. The usual length of the fatal paroxysm is stated by Parry,[1] to be from three to six hours.

The *thoracic* variety of the malady is often combined with the one just described. Its most characteristic trait is violent dyspnœa, caused by overwhelming congestion of the lungs. It is perhaps the most rapidly destructive of all the forms of the disastrous affection.

In the *cerebral* variety the temperature-curve is not that of any special type of malarial fever. The abnormal state of the brain manifests itself either by coma or by delirium. In the former case there is usually preceding stupor with occasional delirium; the pulse is slow and full; the face is dull, and either flushed or livid; indeed, some of the symptoms which are observed in apoplexy show themselves. When, on the other hand, delirium is marked, we have much the

[1] Amer. Journ. Med. Sci., July, 1843.

same morbid phenomena as in acute meningitis; the patient is wild; he sings, he cries. He may die in this state without coma supervening; but a comatose condition generally succeeds rapidly to the fierce excitement. Should recovery take place, the delirium gradually ceases.

Another variety much dwelt upon is the so-called *algid* form. This is not often seen in this country; but is not uncommon in Corsica and Algeria. The disease is more than a mere continuation of the cold stage of a paroxysm: usually the characteristic symptoms manifest themselves during the period of reaction. The pulse slackens, and finally ceases; the extremities, face, and trunk become in succession rapidly cold. There is no thirst; the skin feels like marble; the breath is cold; the voice broken. The mind is clear; the expression of the countenance impassive and like that of a dead man. There may be frequent attacks of syncope; or excessive sweating; or vomiting and choleraic discharges occur. These symptoms go on steadily towards death, unless decided reaction be brought about.

In none of these forms of congestive fever is the first paroxysm apt to be of a pernicious character. In the majority of instances the disease begins as ordinary periodic fever, and it is only in the second or third paroxysm that the alarming symptoms appear. Nor is the first pernicious paroxysm likely to prove mortal; generally it is not until the second or third that a fatal issue is to be apprehended. Proper watchfulness will sometimes detect, even at the onset of the attack,—by the unusual prolongation of the cold stage, or by the irregularity of the pulse, or by the great sensitiveness in the splenic region and by the pain which pressure there may occasion all over the body, or by an imperfect hot stage, or by the feeling of internal heat while the surface is really cold,—the danger that is approaching, and arrest its further steps by the bold use of antiperiodics.

The cause of this desperate disease is a highly active malarial poison, and very likely some peculiarities of the malarial parasites. Should the patient even weather the first attack completely, he is not wholly out of danger; he may have a second seizure quite as perilous within the same season. Dock[1] has recorded in detail the study of a case of pernicious malarial fever characterized by an enormous development of plasmodia in the blood, with consequent anæmia and melanæmia; parenchymatous degeneration and inflammation in liver, kidneys, and stomach; thrombosis in various organs; hyperplasia of the spleen and lymphatic glands. On micro-chemical examination the

[1] Amer. Journ. Med. Sci., April, 1894, p. 379.

pigment in the malarial parasites failed to respond to tests for iron, while deposits in the tissues themselves yielded such reaction.

Hemorrhagic Malarial Fever.—Closely connected with congestive fever, indeed a form of it is that pernicious malady which is known as the yellow disease, icterode pernicious fever, malarial hæmaturia, hemorrhagic malarial fever, or black-water fever. It is the same disease as that which some of the French writers have long described as hæmaturic bilious fever, and is found in intensely malarial places, sometimes in epidemics. It usually occurs in those who have already suffered much from malarial fever, and is almost always ushered in by a marked chill, longer usually and more intense than the patient has had in the preceding seizure of intermittent,—for often the dangerous paroxysm is preceded by one of ordinary kind. Soon after the protracted chill, distressing nausea and vomiting are noticed, as well as headache, great restlessness, and quickly developed, deep jaundice. The fever which follows the chill is not high, the pulse is rarely extremely rapid, the patient is very thirsty. In a few hours after the chill, pain in the right hypochondrium, in the epigastrium, and over the kidneys is encountered, and a dark-colored, bloody urine is voided. Sometimes hemorrhages occur also from the nose and bowels. The type of the fever is either intermittent or remittent, occasionally it is continuous. The bloody urine—for I know the dark-colored urine, from the specimens I have examined, to be bloody or to contain large quantities of dissolved hæmoglobin—is at times associated with considerable albumin and with tube-casts. The parasite is of the æstivo-autumnal form. Baccelli[1] attributes the hæmoglobinuria not to the malarial parasite, but to its toxines.

If the case progress unfavorably, the pulse rises, cold sweats occur, purpuric spots appear on the skin, and the signs of uræmic poisoning are not unusual. In the intermission or remission the symptoms abate considerably, jaundice and bloody urine cease to a great extent, perhaps almost entirely,—at least this is true of the latter symptom,—but they recur in the paroxysms, which may happen every day or every ten or twelve hours.

The disease may prove fatal in three days; but generally it lasts longer. Convalescence sets in slowly, and not until the urine has entirely and permanently cleared. It is thought by several observers, especially by Tomaselli, Ughetti, and other Italians, that the disease is not due to the malarial infection, but to the toxic influence of quinine. But this view is not adopted in this country.

[1] Policlin., Jan. 1897.

As regards the diagnosis of the disease, there are but two diseases that closely resemble it. One is *intermittent hæmoglobinuria*. Now, undoubtedly some of the recorded cases of this are cases of the malady under discussion; but in those to which the name can be fairly given the absence of malarial elements in the blood, of jaundice, of red blood-disks in the urine, and the want generally of fever, supply the distinguishing traits. From *yellow fever*, for which hemorrhagic malarial fever may be mistaken, it differs in the speedy occurrence of marked jaundice, in the bloody urine, in the extreme rarity of black vomit, in the course of the fever with its recurring paroxysms, and in the high degree of malarial poisoning which the history of the case and the examination of the blood proves.

Then, again, the malarial poison may affect the kidneys, producing altered secretion, transitory albuminuria, or even nephritis. Albuminuria was found by Thayer[1] in nearly half the cases of the malarial fevers of Baltimore, and much more frequently in the æstivo-autumnal infections than in the other forms. In this form, too, acute nephritis is more common than in the other varieties. The malarial infection may lead to chronic renal disease. In all these kidney complications the history of the case and the examination of the blood for the malarial parasites are of the greatest importance. The cases of nephritis following hæmoglobinuria are always grave.

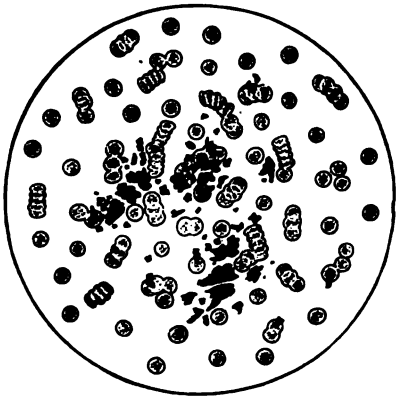
Before proceeding to the discussion of another subject, I shall here devote a few pages to the consideration of some of the irregular forms and modifications of malarial poisoning, and to its share in producing febrile disorders of blurred and uncertain type. Practically, this is of great importance, and specially of importance to American physicians.

In the first place, I shall speak of the *chronic malarial poisoning*, or *malarial cachexia*, so often seen among inhabitants of malarial districts. It manifests itself by lassitude, debility, torpor of the liver, and enlargement of the spleen. The stools are often black, the digestion is impaired, the complexion sallow. Occasionally attacks of jaundice occur, which rather relieve than aggravate the unhealthy state of the system. Sometimes the noxious influence shows itself in another way: the patient is seized with nausea, and with gastric irritability so great that almost everything he takes is instantly rejected. The tongue is coated, the skin dryish; but he has little if any fever. The bowels are confined, the urine is turbid. He is restless, and as weak as if he had typhoid fever; but he has neither an eruption nor

[1] Amer. Journ. Med. Sci., Dec. 1898.

diarrhœa. His sleep is disturbed, and he often suffers with hyperæsthesia of the scalp, and neuralgic pain shooting over the forehead and causing twitching of the eyelids. After remaining from six to seven days in this condition, his nails, perhaps at a certain hour every day, are noticed to become bluish; or he feels chilly, and a slight fever immediately afterwards sets in. The return of these febrile symptoms is checked by quinine, and the patient enters upon a slow convalescence, remaining for a long time enfeebled. Again, there may be headache, coming on at a certain hour, associated with rise of temperature; or attacks of diarrhœa or of vomiting; or a persistent slight febrile state with the temperature from 99° to 100°, with occasional rises. We also encounter malarial diseases of the eye, pulmonary congestion of malarial origin with the parasites in the sputum, malarial aphasias,[1] malarial atony of the bladder,[2] neuralgias, especially of the supraorbital and intercostal nerves, and malarial palsies.

FIG. 82.



A drop of blood taken from the finger of a man the subject of malarial cachexia. The granules of pigment, as well as the larger fragments of irregular form, are seen among the blood-globules. The pigment was for the most part black; some of the particles were reddish brown.

In these, as in a case under my care at the Pennsylvania Hospital in 1889, the detection of the malarial corpuscles in the blood led to the diagnosis of the affection. Indeed, in any of these doubtful and suspected cases, in which, too, the periodicity may ultimately be lost, careful and repeated blood examination is essential. The usual form of parasite found is the æstivo-autumnal; pigmented leucocytes are also not uncommon. But as regards the parasites in all these in-

[1] Longayet, Indian Lancet, Jan. 1897.

[2] Marion, New York Med. Journ., 1897.

stances of chronic malarial infection, they may not be detected except after several examinations.

In the malarial cachexia we have not only the ordinary signs of anæmic blood, and with these frequently enlargement of the spleen, dropsy, and hemorrhagic tendencies, but the blood itself exhibits peculiar signs. It will show not only the malarial parasites, but considerable pigment, the result of the destructive changes in the hæmoglobin of the red corpuscles. Besides the black pigment there is also a yellowish or rusty-colored pigment, the seat of which, however, is more especially the spleen, liver, and bone-marrow. The pigment granules are found not only within the malarial parasite, but also exist free, and, accumulating in the capillaries, produce clogging, with secondary results of disturbed circulation, and altered nutrition in the brain, liver, kidney, or of whatever part the vessels should supply. For the pigment to be of diagnostic value, it must be present in decided amounts; for J. F. Meigs[1] found pigment in the blood of those who had never had malarial fever or had never presented any signs of malarial poisoning. In the malarial blood the number of leucocytes is diminished, with, as Thayer states, a relative increase in the large mononuclear forms.

Typho-Malarial Fever.—Following the observations of Woodward during our civil war, the thought obtained wide currency that there existed a special form of fever, typho-malarial, running a definite course and with characteristic lesions. It was supposed to be a hybrid, generated by the malarial and typhoid poisons, with, in the case of the camp fevers, an admixture of scurvy; and the so-called "Chickahominy fever," seen among soldiers who contracted it in the swamps of the Chickahominy, was its most striking illustration. But the verdict of the profession now is, that there is no such fever as a distinct disease. Yet with our present means of research it can be proved that there is undoubted coexistence of the malarial and typhoid infections. There are malarial cases in which true enteric fever happens, or typhoid-fever cases in which the malarial poison has been held in check by the typhoid infection, and does not show itself until late in the disease; cases beyond doubt clinically, in which the Widal reaction is positive, and malarial organisms are found in the blood. Thompson[2] has reported several such instances of concurrent disease; Lyon[3] has brought together others; and I have

[1] Pennsylvania Hospital Reports, vol. i.
[2] Amer. Journ. Med. Sci., Aug. 1894.
[3] Ibid., Jan. 1899.

records of twelve, one of which was separately published[1] in a clinical lecture, and ten of which were subsequently analyzed. Pathologically, also, a number have been studied in an interesting communication by Muehleck.[2]

Now, it is a question whether, irrespective of the Widal test and the microscopic examination of the blood for malarial elements, such cases can be recognized clinically. Not with certainty. Yet they may be suspected from chills occurring late in the disease, and decided sweating following; from obvious and apparently causeless temperature-rises, and marked irregularity of temperature without such rises; and from long duration of the fever. In all such cases repeated examination of the blood for malarial organisms should be made. The parasites I found were tertian or æstivo-autumnal, and frequently decidedly pigmented; an instance of the quartan type in one of these combined typhoid and malarial fevers has been published by Craig.[3]

There is, then, such a morbid condition as a typho-malarial fever, but not as a separate disease, and not in the sense in which it has been understood. It is a concurrence rather than a blending,—a typhoid fever, after all; and, if we are to give it a name, malario-typhoid would be appropriate.

Eruptive Fevers.

The eruptive or exanthematous fevers form a group having numerous features in common. They are characterized by a period of incubation, during which the poison lies dormant; by a fever preceding the eruption; by an eruption which presents a distinct aspect in each disease, and which pursues a definite, clearly defined course until it, and, with it, the febrile malady, disappears. Moreover, they are all very prone to occasion serious sequelæ; are all, in the main, disorders of childhood; rarely attack the same person twice; and are contagious. These remarks apply particularly to the three chief exanthematous fevers: scarlet fever, measles, and smallpox. In great part, too, they hold good in regard to erysipelas, described here in connection with the eruptive fevers.

Scarlet Fever.—Scarlatina affects both children and adults, and is marked by great heat of skin, frequent pulse, sore throat, and an early scarlet eruption. These symptoms are preceded by an uncertain, generally a short, period of incubation, but soon exhibit their

[1] Philadelphia Med. Journal, May 6, 1899.

[2] Ibid., May 20, 1899.

[3] Ibid., June 17, 1899.

striking features. The febrile excitement is characteristic; the skin is very hot and generally dry, and the rapidity of the pulse so great that often by this sign alone we may, especially in the midst of an epidemic, predict the coming eruption. Vomiting, too, is a frequent symptom at the beginning of the illness. The temperature, which may reach between 105° and 106°, does not fall with the appearance of the eruption. The highest temperature occurs on the second or the third day.[1] The temperature continues high until the eruption is completed and at its height. It slowly declines as this fades, and with the occurrence of desquamation attains the norm; but it may persist, with marked morning remissions and evening exacerbations, when the eruption has gone and during the first week of desquamation.

The *rash* appears on the second day of the disease. It comes out almost simultaneously all over the body, although, on close scrutiny, it may be soonest perceived on the neck and the breast. At first the surface exhibits an almost uniform red blush, which disappears momentarily on pressure, or rather pressure leaves a white stain on the skin, which quickly again reddens from the periphery to the centre. Soon, however, the eruption presents an unequal aspect; it is of more vivid scarlet hue in some parts of the body, as in and around the flexures of the joints, and is not everywhere smooth. Here and there are seen elevated rough points of darker tint, edged by the red integument, and not infrequently vesicles containing a thin fluid. The skin is very hot and itchy, and tumefied, especially on the hands and feet. The eruption declines on the fourth or the fifth day; by the seventh or eighth, the cuticle begins to come away in large flakes. Sometimes the rash, when at its height, recedes and then appears again. In malignant cases it comes out late, and is either pale and indistinct, or dark and livid. In some instances it is wanting. Some years ago, I saw this "scarlatina sine exanthemate" in a lady, who, watching over the sick-bed of her daughter, contracted the disease and went regularly through it, even to its sequelæ of disorder of the kidneys and swelling of the salivary glands, but in whom not a trace of an eruption could be detected.

The *sore throat* of scarlatina is almost as constant and as characteristic as the scarlet rash. It shows early, sometimes before the eruption, and rarely waits until the third day of the complaint. At first the throat-affection consists in a diffused redness extending over the tonsils, palate, and half-arches, and in a swelling of the tonsils:

[1] Hatfield, article "Scarlet Fever," in American Text-Book of Diseases of Children, 1894.

the patient complains of pain in his throat, augmented by pressure and by swallowing, and of stiffness of the muscles of the neck. After a few days, if the disorder be severe, irritating discharges occur from the inflamed surfaces, and patches of false membrane and superficial ulcerations are seen in the fauces. The glands at the angle of the jaw become much tumefied, and, by pressing on the cervical vessels, produce a tendency to drowsiness and stupor. These are grave symptoms; their occurrence, indeed, is indicative of one of the main dangers in these "anginose" cases of the disease.

The false membranes which are developed last about five or six days; they form as well as reform in patches, and are very easily removed. Sometimes they extend to the larynx; but this does not often happen. They contain masses of streptococci, but no diphtheria bacilli, unless there be a true diphtheritic complication. Yet this is a point that is not accepted by all clinicians. The mortality in these mixed cases is much greater.[1] The acid discharges and the decomposing membranes often occasion a most fetid breath.

The *tongue* has a peculiar look. At first it is thickly coated, and its borders only are red; but soon the fur is cast off, and the whole organ becomes very red and its papillæ prominent. After it has presented this appearance for six or eight days, it returns to its normal condition. In bad cases it is extremely dry and of a brownish hue.

There is always marked *leucocytosis* in scarlet fever, and it reaches its maximum in the first few days of the disease; a close relationship exists between the severity of the rash and the number of leucocytes;[2] and the return to normal is always gradual.

In children the disease frequently sets in with convulsions. In truth, cerebral symptoms of one kind or another are not uncommon at all stages of the malady. In some cases of malignant character, the vomiting, the screams, the grinding of the teeth, the occurrence of delirium and insomnia, make the attack look, at the onset, like one of acute meningitis; but the eruption soon sets all doubt at rest, and, even before it is noticed, the great heat of the skin and the extreme rapidity of the pulse point to the source of the mischief. The nervous symptoms in these dangerous instances of the affection do not, however, cease with the eruption; they may last to the end of the malady. Sometimes they are not noticed until late in the disorder, and after the period of desquamation has fully begun; but the convulsions and

[1] Chabade-Roussk. ark. patol. klin. med. ibakt, Feb. 1899, quoted in Medicat. Mart., July, 1899.

[2] Sevestre, St. Bartholomew's Hosp. Rep., 1897.

stupor—for these are the morbid manifestations then more specially encountered—are owing rather to a diseased state of the kidneys that has been induced, than to the immediate effect of the fever poison.

Occasionally some of the larger joints swell up, and present the appearance of subacute rheumatism. The joints are not, however, very painful on pressure, and generally only two or three are enlarged. Endocarditis and pericarditis may be present as complications, but occur also irrespective of articular involvement, as does chorea.

Further complications of the disease are dropsies, renal hæmaturia, pleurisy, local gangrene, œdema of the glottis, neuritis, diphtheria, and profound anæmia. These complications do not usually arise until at or soon after the period of desquamation; sometimes they lead to long-continued disorder, and become thus the most hazardous of the sequelæ. Other consequences of the affection, lasting, it may be, for years after the febrile attack, are a tendency to boils, swelling of the parotid and of the lymphatic glands of the neck, nasal catarrh, diarrhœa, chronic inflammation of the eyelids, and deafness from inflammation extending up the Eustachian tube to the membrane of the tympanum, or from suppurative destruction in the middle ear. Epilepsy is also a sequel of scarlet fever, more cases being consecutive to it than to all other acute diseases combined.[1] Optic neuritis may follow scarlet fever, without organic change in the brain.

Of all these morbid states, *dropsy* is the most common. The effusion of fluid may be caused by the altered state of the blood; but much more generally it is owing to the poison producing an acute desquamative nephritis: albumin, tube-casts, epithelial cells, and sometimes blood, are found in the scanty urine; and we meet with severe headache, great restlessness, and œdema of the face and extremities, as the attending symptoms. Still, notwithstanding these grave phenomena, the majority of the cases recover, and the kidneys are rarely permanently injured.

The dropsy is apt to show itself between the tenth and the twentieth days of the malady. The albuminous condition of the urine may precede it by several days; yet dropsy may happen without albuminuria,[2] and albumin in the urine is not always associated with dropsy. In most cases of scarlatina albumin is found at some period of the disease for a short time and in small quantities.

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[1] Gowers, Diseases of the Nervous System.

[2] Gee, in Russell Reynolds's System of Medicine; also Quincke, Berlin. klin. Woch., 1882, No. 27; Dyce Duckworth, St. Barth. Hosp. Rep., 1883.

The *state of exhaustion* noticeable at the close of the fever and while desquamation is still going on is at times great,—so great that, in young persons especially, the case wears the look of *typhoid fever*. And the resemblance is heightened by the occurrence of diarrhœa associated with a swelling of the solitary and agminated glands. But the signs of desquamation, the sore throat, the enlargement of the cervical glands, and the history of the affection furnish distinctive marks of the utmost value. We must also bear in mind that an erythematous rash like scarlatina occurs at times in typhoid fever preceding the characteristic rose-spots.

The statements that have just been made concerning the diverse complications of the malady are mainly of interest on account of their exhibiting the intricate diagnostic questions that may arise. Of the recognition of the disorder during the febrile stage it is not necessary to say much, as ordinarily it is not difficult. The distinction between it and the other exanthematous fevers will be seen by glancing at the table, to which a place is elsewhere assigned. I shall only here mention, as bearing upon the differences between scarlet fever and *measles*, that cases are occasionally encountered in which the eruption alone is too ill defined to become the sole basis of an opinion, and that then we have to lay the greatest stress on the presence or absence of catarrhal symptoms and sore throat, and on the march of the symptoms. So, too, with reference to *smallpox*. The rash preceding the formation of the pustules may so strongly resemble that of scarlet fever that a scrutiny of all the attending circumstances, and a careful watching of the eruption for at least a day, are requisite for the detection of the true nature of the case.

An erythematous rash, appearing in blotches everywhere except on the face, has been noticed in *laryngeal diphtheria* after the operation of tracheotomy.[1] But it is very irregular, runs a rapid course, and is not followed by desquamation; a point, it may be here mentioned, distinguishing all the forms of irregular rashes happening at times—though very rarely—in diphtheria, from the scarlet fever eruption. As the result of gonorrhœa we may have symptoms of a low fever associated with a cutaneous rash like that of scarlet fever. The history and progress of the case chiefly distinguish this *pseudo-scarlatina*.[2] The same is true with reference to the so-called *surgical scarlet fever*. It shows an eruption that may be like that of

[1] Bericht des k. k. Krankenhauses, Weiden, 1865.

[2] Ballot, Arch. Gén. de Méd., Sept. 1882. The same author calls attention to a puerperal pseudo-rubeola, a false measles, from blood-infection.

scarlet fever, though the throat symptoms and the sequelæ are lacking. It is most likely of septic origin.

Like measles, scarlatina may be mistaken for rubella. But this really resembles measles more closely, and in examining it presently the differences between it and scarlet fever will become apparent.

An affection with several features like scarlatina is breakbone fever, or *dengue*. The points of dissimilarity may be learned by referring to the description of the malady already given. It well also to remember that certain *drugs*, such as quinine may produce a scarlatiniform eruption.

Scarlet fever may go on concurrently with other fevers. It has been observed with typhoid fever, with varicella,[1] and with smallpox.[2]

Measles.—The symptoms precursory to the specific eruption of this affection are fever, watery eyes, frequent sneezing, flow from the nose, and cough; in fact, all the manifestations of an acute coryza or catarrh. To these diarrhœa is in many instances added, indicating a simultaneous irritation of the intestinal mucous membrane. On the fourth day after the beginning of the morbid signs, a rash is perceived on the face and neck; thence it continues to extend, until, in the course of two or three days, the whole body is covered. The temperature during the first day of the disease is generally from 102° to 103°; if higher, the attack is likely to be severe. On the second or third day—usually on the second, when it may be but 98.6° or 99°—it is markedly lower, and it rises again on the evening of the third or on the fourth day to decided fever heat. The temperature does not at once decline with the rash. Indeed, it is apt to go on rising for twenty-four to thirty-six hours; the occurrence of the eruption does not alleviate the febrile symptoms; on the contrary, while it is spreading to the trunk and the lower extremities, the constitutional disturbance lasts, or more generally increases. But as soon as the rash has fully reached its height, the defervescence is rapid; and from the fifth to the seventh day of the disease the temperature sinks until it is but little above the norm. By the ninth day of the disease both fever and rash have left. Frequently then the cuticle comes away in fine scales, and this desquamation is attended with very annoying itching. The patient, now that he is convalescent, shows his illness: he is pale and somewhat emaciated. Often he still coughs, and his eyes are slightly inflamed. These signs are not unusually the last to disappear.

[1] Church, St. Barthol. Hosp. Rep., 1881; Lond. Med. Record, Nov. 1883.

[2] See the cases of Marson, Medico-Chirurg. Transact., vol. xxx.

Paralysis, of cerebral, spinal, or peripheral origin, may occur in the sequence of measles.[1]

Of all the symptoms mentioned, two are, in a diagnostic sense, of pre-eminent importance: the catarrh and the eruption.

The *catarrh* is nearly constant. It is true that a variety of measles is recognized,—"rubeola sine catarrho;" but this is very rare. Generally speaking, the coryza and catarrh decline with the eruption; occasionally, however, they remain for some time after the rash has left. The feature which distinguishes these catarrhal symptoms from those of influenza is the eruption: before this happens, the diagnosis is uncertain, though we may often suspect measles by the look of the face, the greater intensity of the febrile signs, and the knowledge that the disease is prevailing in the community.

The *eruption* is peculiar: it consists of slightly raised red spots, which coalesce and form blotches of an irregular, crescentic shape; between these blotches the skin is of natural color. The eruption disappears first from the face; in other words, it disappears in the same order in which it appears. As it fades, which it does on the third or fourth day of its appearance, it becomes brownish, and subsequently of a yellowish tint. In its earliest stages it is similar to the papulæ of smallpox; and this similarity may be heightened by its being mixed, as it sometimes is, with a few miliary vesicles. But after the first day of the rash there is little room for doubt. In the one case the spots remain; in the other, they change into pustules.

A very valuable contribution to the diagnosis of measles, especially to its early diagnosis, has been made by Koplik.[2] He has pointed out that from three to five days before the outbreak of the eruption, as well as to be seen afterwards, are found, when the mucous membrane of the cheeks and lips are examined by strong daylight, and limited to them, small, irregular, bright-red spots with a minute bluish-white centre. These spots are most frequent opposite the lower molar teeth, and they are not met with in any other exanthem, or in any disease of the skin.

A question may sometimes arise as to whether the eruption be that of *typhus fever* or of measles. Both are coarse, both often not unlike in color, and both may be developed about the same time. Generally speaking, however, the eruption of typhus fever shows itself several days later than the rash of measles; and, although

[1] Allyn, Medical News, Nov. 28, 1891, p. 617; Carpenter, Medical News, Feb. 13, 1892, p. 183.

[2] New York Med. Record, April 9, 1898.

coarse, it is not crescentic, and is found on the trunk and extremities and only rarely on the face. Moreover, the physiognomy, the excessive prostration of strength, and the marked cerebral symptoms of the low fever are such as to render a differential diagnosis seldom difficult. From hemorrhagic measles the distinction is more difficult; but here, too, the absence of cerebral symptoms is of much importance.

Measles is usually met with in children; but it may be encountered in adults, especially among soldiers, and is in adults a much more severe complaint than in children. In the latter it is not an alarming disease. Only occasionally does it occur in epidemics which present a malignant character. Its greatest danger commonly consists in the eruption disappearing prematurely or appearing but partially, and in the severity of the thoracic complications. These are either acute bronchitis or acute pneumonia.

Acute bronchitis may occur at any period of the disorder, and involve the finer tubes. But it does not generally set in with severity until the eruption has reached its height or is beginning to fade. In young children, symptoms of inflammation of the larynx, or of croup, are at the same period apt to manifest themselves. Acute pneumonia, too, either croupous or broncho-pneumonia, the latter much more often, is met with at this stage of the malady, or sometimes even after convalescence has apparently begun.

Occasionally the thoracic affection leaves a chronic bronchial disease, or a persistent cough and night-sweats point to tuberculosis. It may be, in individual cases, extremely difficult to decide with which of these morbid states we have to deal, and as the physical signs of tubercular consumption are, in children, notoriously ill defined and untrustworthy, we may be obliged to depend upon the presence or absence of tubercle bacilli before coming to a definite conclusion.

An affection formerly very common, *miliary fever*, would be also a source of much confusion were it in our day often encountered. But epidemics of miliaria are now extremely rare. Yet we know that it is a disorder with a prodromal stage of two or three days, during which great irritation of the skin, debility, and a feeling of suffocation are usual. The marked disease begins with profuse sweating and with severe fever, and præcordial and epigastric distress. These symptoms last until the appearance of the rash, generally on the third or the fourth day, though sometimes not until much later, and then, as a rule, slowly subside. The rash appears first upon the neck and the breast, and consists of numerous round or irregular spots, in the centre of which vesicles arise that finally burst and form crusts. The disease ends with desquamation, and generally in a slow convales-

cence. The sweating, the oppression and præcordial pain, and the peculiar eruption distinguish this epidemic disease from measles.

Rubella.—The most striking resemblance to measles is furnished by rubella. This, called by the Germans *Rötheln*, and often spoken of as "German measles," is not a hybrid of measles and of scarlet fever, but a special exanthem, which occurs in epidemics. It displays a red eruption, ushered in by a chill, followed by slight fever, which is accompanied by coryza, cough, and sore throat. The fever lasts for two or three days prior to the eruption, but this is far from constant; indeed, it often does not last more than half a day, or it may be of a week's duration.[1] The temperature rarely exceeds 102.5°. The rash may come out all over at once, or spread in a day or two over the body; it generally appears first on the face and neck. It is most distinct on the face, the scalp, the neck, and the trunk, being more scattered on the extremities: it is specially distinct about the mouth. It first resembles measles, but the spots are round or oval, and smaller and paler, and they soon run together in irregular patches, unlike the well-defined crescentic eruption of measles; they show no tendency, however, to become generally confluent. The patches are of variable size, and, unlike the rash of scarlatina, are surrounded by healthy skin; small spots range themselves around the large ones. They are of deepest color in the centre, but not bright-colored as in measles, nor of the dark red of severe scarlatina, are elevated, and very much influenced by pressure. The eruption lasts ordinarily four or five days, but in severe cases eight or ten. It gradually fades, but it may happen that it fades on the face before it has fairly come out on the legs, and desquamation may ensue, though the scales are small, and never in size like those of scarlet fever. During the continuance of the rash, which is attended with much itching, the general symptoms are greatly aggravated, except the fever, which indeed may be perceptible only at the beginning of the affection; the sore throat and catarrh may be severe, and attended with hoarseness and with inability to swallow; there are congestion of the conjunctivæ and pain in the eyes. Osborn has called attention to enlargement of the small glands at the edge of the hair on the postero-lateral sides of the neck as a pathognomonic sign.[2] As the rash fades, the other symptoms subside. Swelling and even suppuration of the cervical glands are not uncommon sequelæ.

The disease may be very difficult to distinguish from measles,

[1] Edwards, article "Rubella," in Keating's Cycl. of Diseases of Children.

[2] Weekly Med. Rev., Dec. 24, 1887.

except when it is epidemic and affects those who have already had measles. The more sudden onset, often almost feverless, the milder course of the complaint, and the peculiarities of the eruption already spoken of, are guides in separating individual cases. But the appearance of the rash may be ill defined and very misleading. The following table exhibits the differences between well-marked cases of rubella, measles, and scarlet fever:

Rubella.	Measles.	Scarlet Fever.
Period of incubation from nine to twenty-one days; usually eighteen days.	Period of incubation from seven to fourteen days.	Period of incubation from a few hours to seven days; rarely beyond five days.
Premonitory symptoms often wanting, but frequently sore throat. If attack severe, loss of appetite and drowsiness for twenty-four hours before eruption.	Premonitory symptoms common, such as lassitude, loss of appetite, headache, vomiting, watery eyes, catarrh, cough.	Premonitory symptoms; usually feeling of lassitude for a few hours, frequently vomiting. If attack slight, patient complains only of sore throat.
Eruption is mostly the first symptom; dots, rosy-red, with well-defined edges, first behind the ears, on scalp and face, around mouth; extends to neck and chest; gradually covers entire body. Dots coalesce and form patches.	Eruption appears on fourth day; first behind ears, then on scalp and forehead; spreads all over face, body, and limbs, forming crescentic blotches. Eruption is papular in character and dark-red in color; never bright rose-red.	Eruption diffuse, dusky red with interspersed raised spots; appears early about clavicles and on chest, and on covered parts of the body; intensely hot to touch.
Fauces look dry, with a dark mottled red hue; little relation of appearance of fauces to extent of rash. Sore throat may disappear, to recur in last stages of the disease.	Fauces red and swollen throughout activity of the disease.	Fauces vary in appearance from slight to intense dusky redness, with marked swelling, and sometimes with white spots of inspissated secretion; intensity bears direct relation to skin-eruption. Sore throat throughout disease.
No diazo-reaction in urine.	Diazo-reaction.	
Eyes pink-red and suffused.	Eyes red and watery; photophobia.	Eyes unaffected.
Lymphatic glands generally enlarged, tender, hard, notably the posterior cervical, the axillary, and the inguinal.	Not usually affected; the posterior cervical rarely and slightly; bronchial glands always enlarged.	Lymphatic glands of throat and neck at first scarcely discernible, but subsequently enlarged.

Rubella.	Measles.	Scarlet Fever.
Catarrhal symptoms and cough inconstant; there may be a little flaky desquamation; frequently none.	Catarrhal symptoms and cough constant; a little flaky shedding of the epithelium, varying according to the intensity of the rash.	Catarrhal symptoms and cough absent, or slight throat cough. Desquamation in proportion to the extent of the eruption; begins as this fades, and continues for weeks; marked about hands and feet.
Kidneys rarely affected; may be transient trace of albumin.	Kidneys not affected.	Kidneys often implicated; albuminuria; acute nephritis common.
No diarrhœa.	Diarrhœa frequent.	Diarrhæa not uncommon.
Patient, as a rule, does not feel ill.	Usually feels illness much.	In slight cases light illness; in severe cases grave illness.
Tongue clean or slightly furred.	Tongue slightly furred.	Tongue coated with a thick, white fur, peeling from the tip and edges on the fourth day, leaving the "strawberry" tongue.
Pulse slightly accelerated; maintains ratio to temperature.	Pulse usually accelerated; maintains ratio to temperature.	Pulse greatly accelerated, and rapid out of proportion to temperature.
Temperature varies between 102° and 103°.	Temperature usually from 101° to 103°.	Ranges from 103° to 106°; proportionate to rash, but not to pulse.
Infectiveness lasts from ten to fourteen days if disinfection efficient.	Infectiveness does not last for more than from fourteen to twenty days, if disinfection efficient.	At onset only slightly infective; is very infective after first forty-eight hours; infectiveness may continue for six or eight weeks or longer.
Sequelæ, few and not frequent; glandular enlargements may follow.	Sequelæ, bronchitis, pneumonia, pleurisy, ophthalmia, otitis.	Sequelæ, nephritis; enlargement or suppuration of submaxillary and lymphatic glands; otitis; arthritis; endocarditis; epilepsy.
Usually complete recovery in two weeks, or less.	Usually complete recovery in two weeks; is sometimes followed by prolonged period of ill health.	Usually complete recovery; sometimes prolonged convalescence from sequelæ; mortality high in the very young.

Typhus fever, at least as regards the eruption, has some similarity to German measles. But the severe fever, the far greater gravity of the constitutional symptoms, the rash not appearing on the face, and

the absence of catarrhal symptoms, render it strikingly unlike the latter affection.

Rubella is contagious, and affects especially children; it is extremely uncommon after forty years of age. Second attacks are also very rare. It does not protect from either scarlet fever or measles, nor do they from it.

Smallpox.—Smallpox, or variola, attacks both children and adults. It is a highly contagious malady, spreading rapidly among those who are unprotected by vaccination. The period of incubation is generally about twelve days.

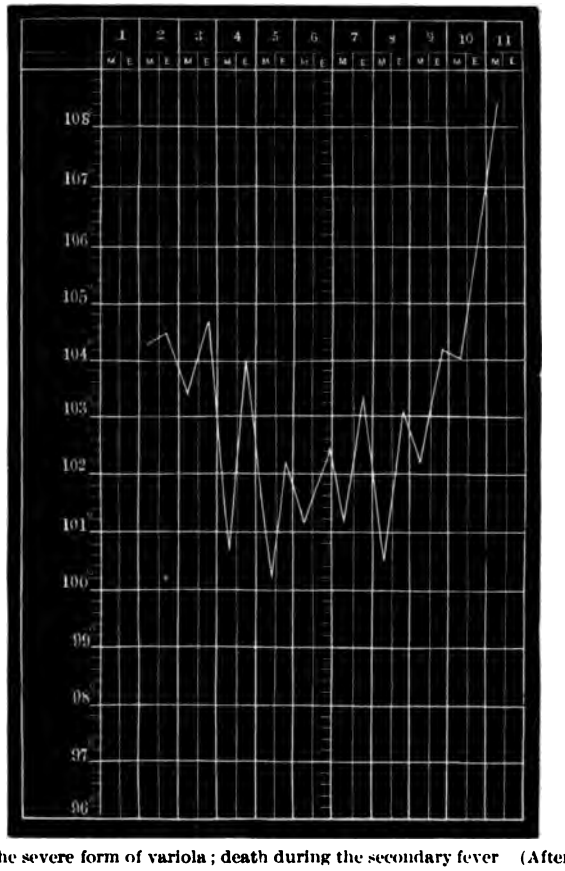
The chief symptoms of the stage of *invasion* are chills, fever, vomiting, pain in the back, and, in children, convulsions. The fever runs high, and exacerbates markedly towards evening; the temperature may reach 106° or more. The pain in the back is severe, particularly in grave cases; it may be attended by pain in the limbs like those of rheumatism; there are also intense headache and restlessness. All these symptoms subside with great relief at the end of the third or on the fourth day, when an eruption shows itself on the lips and forehead and wrist, soon extends to the trunk, and from the trunk all over the body; with the appearance and the spread of the eruption there is a gradual but very decided fall in temperature, often to 100.°

At first the *eruption* has the appearance of papulæ; but on the second and third days the coarse spots undergo a decided change. At the top of each papule appears a vesicle, which gradually becomes larger, and fills up with a thick, milky fluid; in short, becomes a pustule. By the fifth or sixth day, the change has been fully accomplished, and the pustules are spheroidal and lose the umbilicated look which they had while forming. During all this time the temperature does not again rise; the tongue is coated and swollen. On the eighth day pus begins to ooze from the edges of the pustules, and a secondary fever sets in, lasting for three or four days,—until, indeed, all the pustules are broken; this secondary fever is sometimes ushered in by a chill; it is of remittent type, and the evening temperature marks between 103° and 105°. There is gradual and protracted defervescence; crusts form where previously there had been pustules; and as these crusts dry and fall off, the skin beneath is seen to be of a red color, that only slowly fades, and here and there are noticed those scars and pits which the patient carries during the remainder of his life.

Preceding the characteristic eruption in smallpox a red rash like that of scarlatina may be noticed in the pubic and the inguinal or lateral thoracic regions; and at times a very misleading rash of measly form.

When the pustules are in great abundance, they run together, constituting *confluent* smallpox. The eruption may be discovered a day earlier than in the discrete form, and the rough, red blotches are often so thickly clustered as to give a uniformly red aspect to the whole surface. When the pustules completely fill up, whole portions of the face or of the trunk seem to be covered by one extensive

FIG. 83.



Temperature in the severe form of variola; death during the secondary fever (After Wunderlich.)

pustule, which gradually dries into a continuous brownish and most disfiguring crust. While the process of maturation is going on, the features are observed to be greatly swollen; the eyes may be hidden from view; the nose and lips are tumid; conjunctivitis is not uncommon. The patient complains of the tension of the skin, and not infrequently of sore throat and of a steady flow of saliva from the mouth,—a symptom that may be also met with in measles. The sec-

ondary fever is violent, far more so than in discrete variola. It may not appear until a day or two later, but lasts longer, shows a higher temperature, and is the period of danger, since it is at this time that death is most apt to happen. Before death the temperature is sometimes extraordinarily high, 108° or upward.

A fatal issue is often preceded by a dry tongue, by delirium, and by great restlessness; by what, in fact, are called typhoid symptoms. Sometimes death is occasioned by attacks of dysentery or of diarrhœa, by inflammation, œdema or necrosis of the larynx, extensive pharyngitis, by acute endocarditis, or by plugging of a vessel in the brain. A case of variola has been reported complicated, during convalescence, by convulsions, followed by left hemiplegia, in which after death an area of softening was found in the motor area of the right cerebral hemisphere, due to vascular occlusion.[1] Cases of variola have also been observed presenting peripheral neuritis or purulent peritonitis.[2] Other complications, not infrequently fatal, are pleurisy and broncho-pneumonia. Sometimes the patient sinks at the onset of the disease. In these *malignant* cases, mostly met with at the beginning of an epidemic, he dies from the virulence of the poison. He is stupid, delirious; the eruption seems, as it were, to struggle to reach the surface, is ill defined and of a livid hue, and may fail to appear until after death. Many of the malignant cases, too, are of the hemorrhagic type, marked by petechial blotches and ecchymoses, and profuse hemorrhages from mucous membranes. The specific microorganism of smallpox is still undiscovered.

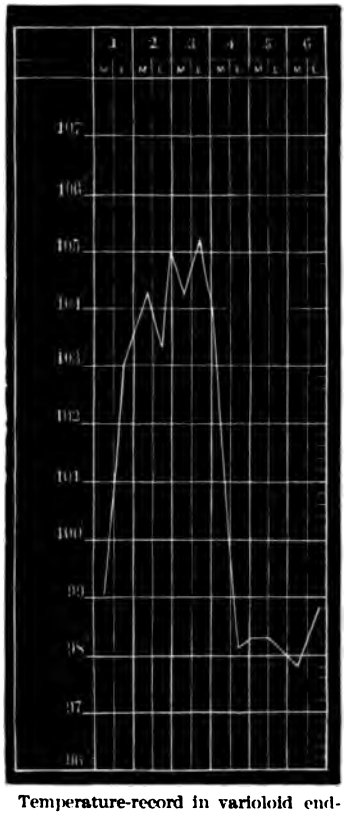
The sequelæ of smallpox are chronic diarrhœa, glandular enlargements, boils, various diseases of the eyelids and eyeballs, otitis media, and suppurative arthritis. Smallpox is occasionally met with during the progress of other disorders, blending its symptoms with those of the complaint to which it becomes superadded. It is thus found as an intercurrent affection in typhoid fever, in typhus, in scarlet fever, and in measles; yet even then there is no difficulty in recognizing its peculiar traits,—its lumbar pain and characteristic eruption. Ordinarily the detection of variola is extremely easy, except at its onset. But the points of similarity it may present, in its early stages, to typhus fever, and to several other diseases, have been already discussed, and need not be repeated; we have often to wait the course of the eruption before framing a positive diagnosis from the symptoms alone, and without taking into account the epidemic influences pre-

[1] Davezac and Delmas, Journal de Médecine de Bordeaux, 1893, No. 38, p. 421.

[2] Auché, Bulletin Médical, Jan. 25, 1893.

vailing. When the disease is fully developed, all difficulty in its diagnosis ceases. In the period of invasion the pain in the loins is the most significant differential sign. It is by this alone that we may be enabled to tell the scarlatiniform rash or the measly rash that is sometimes found to precede the papules of smallpox; though these initial rashes are generally much more localized and not so widely diffused as those of real scarlet fever or measles, and the bastard scarlatina has not the vivid hue of the true disease, nor the measly rash the coarse ness and hardness of the papule of smallpox.

FIG. 84.



Temperature-record in varioloid ending in recovery: the absence of secondary fever is clearly seen. (After Wunderlich.)

The contagion of smallpox does not always manifest itself by an attack of variola. Sometimes it is modified by happening in a person who is partially protected by vaccination. This *varioloid* disease is mild and very rarely fatal; it protects against smallpox. It is distinguished from variola by the pustules passing more quickly through all their stages, and, above all, by an absence of secondary fever. Soon after the eruption—within thirty-six hours—the thermometer shows freedom from fever, and, unless serious complications happen, the temperature remains nearly normal. The suppuration is far less deep; and the resulting cicatrices are often scarcely discernible.

Varicella.—A specific disorder similar to but not identical with variola or varioloid is chicken-pox, or varicella. It differs, as regards its symptoms, from smallpox in the leniency of the introductory fever; in the eruption beginning generally first on the trunk, occurring often on the second day, though it may not show itself until the end of the third, and continuing to appear and disappear in crops, the mass of the eruption, however, having become evident within twenty-four hours; in the vesicles being surrounded by little or no inflammatory redness; in their remaining vesicles and not becoming pustules; in their attaining their height on the third or fourth day of the eruption,

of febrile invasion; in all, too, although the eruption takes its origin at one spot, and generally on the face, it is not limited there. The thickly-clustered blotches of beginning *confluent smallpox* give at times to the face the look of erysipelas. Yet here, also, evidences can be found of a rash about to appear all over the body; and doubt is soon dispelled by the progress of the eruption. Sometimes vesicles and even irregular pustules form in erysipelas, and the malady may be looked upon as a chronic disease of the skin, such as eczema, pemphigus, or impetigo; but these affections lack the history of a recent acute disease, and in reality the likeness is not a striking one. The closest similarity is to herpes zoster of the forehead and face. But the eruption in this does not pass the middle line.[1] The red color of the skin, the fever, and the absence of colic and of gastro-intestinal attacks, distinguish erysipelas from the transient but recurring swellings of *angeio-neurotic œdema*.

Erysipelas may break out in one part of the body after another and the disease be thus kept up for a long period. This *erysipelas migrans* runs its course more rapidly and completely in one part than in another, and in accordance with a general law which it obeys.[2]

Erysipelas may be confounded with *mumps*. The error is mainly caused by stress being laid on the redness which is frequently found beneath one or both ears in parotitis, but which, unlike erysipelas, is attended with much pain on moving the jaw, and with decided glandular tumefaction. The redness, moreover, shows no tendency to spread, and rarely continues for the four or five days during which mumps lasts. In very young children, however, there may be some difficulty in diagnosis. I have seen the glands at the angle of the jaw swollen for one or two days prior to the discoloration over them taking on an erysipelatous blush, which then spread rapidly, and became associated with swelling of the glands of the other side. The glandular complaint was the complication of erysipelas.

A fever with a distinct pharyngitis as a local manifestation, the so-called *pharyngeal fever*, is probably an epidemic erysipelatous fever of light type. It has been particularly described by Austin Flint, Rochester,[3] and Harvey E. Brown.[4] The fever lasts from three to six days, and, besides the marked pharyngitis, is attended with swelling of the lymphatic glands of the neck, accompanied by pain. The disease shows a proportion of cases with erysipelas of the face.

[1] Fagge, Practice of Medicine, vol. i. p. 271.

[2] Traced by Pflüger in 70 cases; quoted in Schmidt's Jahrb., No. 7, 1873.

[3] Buffalo Medical Journal, 1857.

[4] Flint's Principles and Practice of Medicine.

CHAPTER XIII.

DISEASES OF THE SKIN.

To facilitate the discrimination of diseases of the skin, they have been grouped into classes. An extensively used system of classification takes for its basis the anatomical seat and arrangement of the cutaneous malady: it is that of Hebra. As developed by him, it is, however, not a purely anatomical, but a mixed system, resting largely on a pathological basis. Similar is the classification of the American Dermatological Association. All diseases of the skin are arranged in eight classes: Disorders of the Glands, sweat and sebaceous; Inflammations; Hemorrhages; Hytertrophies, of pigment, epidermal, and papillary layers, and of connective tissue; Atrophies, of pigment, hair, nail, and cutis; New Growths, of connective tissue, vessels, and granulation-tissue; Neuroses; and Parasitic Affections, vegetable and animal. Whatever classification we adopt, when a disease of the skin is presented for examination we generally first endeavor to ascertain the special group it belongs to; for instance, is it macular, papular, vesicular, or pustular, or does it present lesions representing more than one group? Having determined this, we next fix which member of the group it is, and then regard its precise seat, and its pathological causation. When this has been accomplished, we inquire into the history of the affection and its duration, whether acute or chronic; take into account the general condition of the patient; search for the evidences of a cachexia or of some visceral disturbance,—a study the importance of which is as great as that of the recognition of the cutaneous malady; and trace, as far as possible, the cause of the disorder. In many instances microscopical and bacteriological examination will be necessary to supplement the clinical evidence and complete the diagnosis.

Here is a table in which cutaneous affections, omitting some of the less important ones, are grouped according to their most obvious features, as well as according to their pathological bearings:

Diseases of the Skin.

- **Inflammatory**
  - Erythematous Diseases
    - Initial rashes of eruptive fevers.
    - Erythema.
    - Roseola.
    - Urticaria.
  - Papular Diseases
    - Papular eczema.
    - Lichen.
    - Prurigo.
  - Vesicular Diseases
    - Eczema.
    - Herpes.
    - Dermatitis herpetiformis.
  - Bullous Diseases
    - Pemphigus.
    - Hydroa.
  - Pustular Diseases
    - Acne.
    - Boils, or Furuncle.
    - Sycosis non-parasitica.
    - Impetigo.
    - Ecthyma.
    - Rupia.
    - Glanders.
  - Squamous Diseases
    - Psoriasis.
    - Pityriasis.
    - Ichthyosis.
    - Squamous eczema.
- Maculæ; Pigmentary Changes
  - Melasma.
  - Ephelides.
  - Vitiligo.
  - Chloasmata.
  - Nævi.
  - Purpura simplex.
  - Xanthoma, or Xanthelasma.
- Hypertrophies of Special Textures
  - Elephantiasis Arabum.
  - Scleroderma.
  - Keloid.
  - Dermatolysis.
  - Warts, Corns, etc.
- Atrophies
  - As of the Hair; the Nails.
  - Senile Atrophy.
- Parasitic Diseases
  - Scabies.
  - Phtheiriasis.
  - Favus.
  - Anthrax.
  - Tuberculosis.
  - Molluscum epitheliale.
  - Lepra.
  - Mycetoma.
  - Actinomycosis.

DISEASES OF THE SKIN.—*Continued.*

- PARASITIC DISEASES
  - Tinea sycosis, or Mentagra.
  - Tinea circinata.
  - Tinea tonsurans.
  - Tinea decalvans.
  - Tinea versicolor, etc.
- NEW GROWTHS
  - Cancer.
  - Sarcoma.
  - Molluscum fibrosum.
  - Lupus.
  - Leprosy, etc.
- ALTERED GLAND-SECRETION
  - of Sebaceous Glands
    - Seborrhœa.
    - Comedo.
    - Sebaceous cyst.
  - of Sweat-Glands
    - Hyperidrosis.
    - Anidrosis.
    - Chromidrosis.
    - Bromidrosis.
    - Miliaria, etc.
- NERVOUS AFFECTIONS
  - Hyperæsthesia.
  - Anæsthesia.
  - Pruritus.
  - Neuroma.
  - Dermatitis herpetiformis.
  - Herpes zoster.
  - Peliosis rheumatica.
  - Plica polonica.
  - Alopecia areata.
- CONSTITUTIONAL SKIN AFFECTIONS
  - Syphilodermata.
  - Scrofulodermata, etc.

Most diseases of the skin are again subdivided into several varieties, based, for the most part, on their duration, situation, form, feel, and color. Thus, we have constantly recurring the terms fugax, inveterata, capitis, facialis, palmaris; guttata, when like a drop on the skin; nummularis, when like a coin; larvalis, like a mask; the qualifying words læve, induratum; circinatum, annulatum, marginatum, indicating configuration, and the adjectives of color, nigrum, rubrum, versicolor. But these divisions are all of secondary importance; and in this outline not much regard will be paid to them. Premising this statement, let us briefly examine the characteristics of the various cutaneous affections of more common form, beginning with those of inflammatory origin.

Erythematous Diseases.—There are only three affections which, strictly speaking, come under this division of cutaneous complaints: erythema, roseola, and urticaria. In all of these the skin is

more or less red, and its surface unbroken; the hyperæmia affects chiefly the papillary layer.

Erythema.—This is characterized by a uniform and continuous redness of the skin, occurring in irregular patches of some size, attended with burning, and with but slight, if any, swelling, and disappearing without desquamation or mark or scar. The eruption is chiefly found on the back of the hands, the forearms, the legs, and the face and neck; rarely on the trunk. There is little or no itching. The affection may be due to the action of heat or cold, or of irritants; or it may be connected with some visceral abdominal disorder. It is usually acute. There is only one variety apt to be combined with decided constitutional or febrile symptoms,—the hard, painful, reddish protuberances most commonly seen on the legs, and constituting the so-called *erythema nodosum*. This form of the complaint, in which there is a serous effusion, is chiefly observed in those of rheumatic diathesis, and, unlike the simple erythema and the erythema intertrigo, which are mere hyperæmias, is classed with the exudations or inflammations. All the exudative forms of erythema may be grouped under the title of *erythema multiforme*, the varieties of which are the papular, bullous, and nodose, the lesions appearing principally on the backs of the hands and feet.

There is a *desquamative form of erythema* resembling scarlet fever, attended with fever of a few days' duration, with epistaxis, and showing an extraordinary tendency to relapse. The eruption is uniform and intensely red, and there is no sore throat, or there is mere redness of the fauces. *Erythema solare*, or superficial dermatitis following exposure to the sun's rays, is usually followed by free desquamation.

A chronic form of erythema results from pressure, or the rubbing together of folds of skin, the *erythema intertrigo;* a slight discharge may coat the rubbed surface. It is liable to acute exacerbations.

Roseola.—This term is applied to circumscribed spots of a rose-red color and of a more or less circular form. The spots are smaller than those of erythema simplex. In *erythema congestivum*, or roseola, there is slight fever, and at times redness of the fauces. The affection often exists in connection with a derangement of the stomach, or with rheumatism, is frequent in summer and in autumn, is generally acute, and bears a certain resemblance to scarlatina and to measles; but it is not contagious, its constitutional symptoms are much milder, the rash is rosy, not crescentic, nor present over the whole body, and we find neither the marked sore throat of scarlet fever nor the catarrh of measles. A rose-rash occurs in the course of typhoid fever, and there is also a syphilitic form.

Urticaria.—Nettle-rash gives rise to prominent and perfectly smooth patches, the color of which is either redder or whiter than the surrounding skin; the white wheals may be surrounded by a red border. The wheals are generally small, but they may be of the size of the palm. The eruption is fugitive and capricious, is attended with more itching, burning, and tingling than the other exanthemata, and is much more evanescent, generally disappearing in two days at farthest. It may, however, exist in a chronic form, the wheals coming out in constant succession, especially after scratching or other irritation of the surface. Pigmentation occurs in the variety known as *urticaria pigmentosa.*

The cause of urticaria is irritation of the gastro-intestinal, pulmonary, or urinary mucous membrane. Certain kinds of fish, especially shell-fish, are particularly prone to produce it; so do mushrooms and strawberries. At times it is due to menstrual disorders, or to sudden emotion, or to the excessive use of mineral waters, or to antipyrin. It may be secondary to the itch, or to phtheiriasis. It occurs in cerebro-spinal fever, and is common in dengue, especially in children.[1]

Urticaria is most probably a reflex phenomenon, caused chiefly by reflected irritation to the cutaneous vasomotor nerves. Urticaria resembles erythema nodosum; but there is no itching in the latter affection, which is chiefly found in the lower limbs, and the swellings change like bruises.

Papular Diseases.—A papule, or pimple, is a small elevation of the cuticle with an inflamed base; it does not contain fluid, and usually terminates in desquamation. It results from a small amount of lymph or a newly formed growth in the derm itself.

Lichen.—This furnishes the best-marked example of a papular eruption. It consists of minute conical papulæ, generally of reddish color, and occurring in clusters. It is most frequently encountered in the summer months and in adults, and often in persons who have been exposed to much fatigue or anxiety. Sometimes it is evidently connected with disordered digestion. It is usually chronic. There is often a mixture of papulæ with an eczematous eruption. Prickly heat, or lichen tropicus, frequently exhibits also sudamina.

In the *lichen ruber* of Hebra the red papules are of the size of the head of a pin; they spread by peripheral growth, are flat, irregular, and have a glazed look and very slight scales; there is considerable itching. The disease, which is an inflammatory one, is chronic; its common site is on the forearm. It resembles psoriasis, but at the

[1] J. C. Wilson, Treatise on the Continued Fevers, 1881.

edge of the patch are the characteristic papules. Poor nutrition and nervous exhaustion are its main causes.

In the *lichen scrofulosorum* the eruption consists of little pale papules, which are chiefly found on the trunk. There is no itching; but we find marked signs of scrofula.

The lesions of *lichen planus* are small, hard, red papules, that may be umbilicated and coalesce into patches. In the latter case scaling occurs, and more or less itching is a frequent accompaniment. The smallness of the recent lesions, which are at first of the same color as the surrounding skin, the flat glazed tops of the older papules, the pure white color of the silvery scales, which are not heaped up, and the unsymmetrical character and distribution of the patches, none of which are circular, will serve to distinguish this from *psoriasis*. *Papular eczema* has its lesions in groups upon an inflamed base, and vesiculation and desquamation occur.

Prurigo.—This is characterized by a papular affection of the skin attended with excessive itching. It is a very rare disease in this country.[1] The pimples are generally torn by the finger-nails, and are surmounted by black scabs. They are not red, as those of lichen usually are, and are, as a rule, larger, and accompanied by much more pruritus and by thickening of the skin. The affection may or may not be attended with constitutional symptoms. It is very obstinate, especially when happening in old persons. It generally affects the legs, the arms, and the trunk, rarely the face and the neck, never the palms and the soles. The skin of the anterior and outer part of the leg is most changed: that over the flexors in the forearm is always healthy. The distressing disorder may be purely local, occurring around the anus, or on the scrotum and the root of the penis, or on the pudenda. Some of these cases, however, though called prurigo, present no papulæ, and the disorder, is due to perverted sensibility of the cutaneous nerves alone, and is really a pruritus. Prurigo is often attended with eczema.

Many supposed instances are not really prurigo, but phtheiriasis, due to the irritation of body-lice, that produce papules, whose apices are scratched off and show little points of dried blood. True prurigo is frequently found to be connected with deterioration of the health, and is chiefly met with among the poor and the neglected. It may last a lifetime, beginning in childhood. Its local forms are associated with irritation of the bladder, the rectum, or the uterus.

Papules and tubercles, or large papules, occur in the latter stages of

[1] Only 34 cases in 123,746 of skin-disease: Von Hardtmann, on Skin-Diseases.

syphilis; they are often preceded by the pigmented erythematous syphiloderm. Gumma is a tertiary manifestation, mostly appearing in the subcutaneous or submucous connective tissue without inflammation, irritation, or itching, the lesions ultimately attaining a considerable size. At first the color of the skin is not changed, but finally it becomes deeply congested and glazed, and as the contents of the lesion soften, the overlying skin breaks down, and the purulent material is discharged.

Vesicular Diseases.—These are characterized by an effusion of a clear or a sero-purulent fluid beneath the epidermis, which is generally raised in small elevations. To the class of vesicular diseases belong especially eczema and herpes.

Eczema.—The malady consists of minute vesicles collected together in irregular patches. The vesicles are often confluent, and it then appears as if the whole surface were secreting fluid. This may harden, from exposure to the air, in scabs of various thickness and color. The skin itself is often of a vividly red hue; indeed, it is inflamed, and a new cell-growth takes place both in the rete mucosum and in the papillary layer of the derm. It is there that the effusion of serum begins. In chronic cases the inflammatory infiltration extends deeper.

Eczema is the most common of all the cutaneous maladies; but it is not contagious. It may affect the whole body, yet is ordinarily limited to some portion of it. It is acute or chronic. The former is generally seen as the effect of local irritants, and may be met with in young and healthy persons. Chronic eczema is more usual, is often the consequence of constitutional disturbance, and is frequently found to be associated with some disorder of the digestive system. It has as a frequent seat the flexor surfaces of the limbs. Dentition and unhealthy milk are common sources of the affection in very young children. In them the disease is extremely apt to attack the scalp and face, forming the complaint often described as "crusta lactea;" or if the secretion be partly purulent, or early become so, and dry into large, dark scabs, the malady is designated as *eczema impetiginodes*. This is most often met with in scrofulous subjects. There is less heat and itching than in other forms of eczema. Eichhoff holds many cases of eczema to be of parasitic origin.

In some of the forms of eczema, especially in its chronic varieties, the vesicles supposed to characterize the disorder can often not be found. This and other reasons have caused several dermatologists, especially Hebra and Anderson, to deny that eczema need be vesicular at all. Infiltration of the skin, exudation on its surface, the formation of crusts, and itching, are held to be its distinctive signs while the

eruption is at its height; but the eruption may consist of clusters of papules, vesicles, or pustules, or there may not be a vestige of any of these, the skin being thickened, red and smooth, and secreting a sticky fluid, or covered with green or gummy crusts, or fissured with deep cracks; yet there are no ulcerations. Not infrequently the disorder begins as an erythema. A scaly form of eczema, *eczema squamosum*, is apt to be confined to the hands and feet. In all the forms of eczema there is severe itching. This itching is especially violent in the form with the deep-red and weeping surface, the *eczema rubrum*, often seen in gouty or in dyspeptic subjects, and having a predilection for the flexures of the joints.

Eczema, when it affects the scalp and face, must not be confounded with the morbid secretion from the sebaceous follicles that gives rise to soft crusts. *Seborrhœa* by preference attacks the parts mentioned; but its crusts, as Hardy has shown, are unlike those of eczema in the readiness with which they are detached and are susceptible of being moulded between the fingers. The surface beneath the crusts, too, is dissimilar. It has an oily, glistening look; there is no discharge. Unna[1] has distinguished a seborrhœic form of eczema, which, beginning usually on the scalp, spreads to other portions of the cutaneous surface; but he attributes the source of the fatty scales and crusts to disorder of the sudoriparous, rather than to the sebaceous glands. Patches of seborrhœic eczema are also found in the sternal region, which, after the scalp, is the locality most frequently affected, the patches spreading by small papules at the border, leaving the centre less scaly and even smooth, while the margin is a red, scale-covered wall.

Eczema may be confounded with *pityriasis rubra*. But this rare disease speedily involves the whole surface of the body, is very chronic, is not accompanied by discharge, and there are large, thin epidermic scales.

Herpes.—This is a vesicular affection, differing from the vesicular form of eczema by the larger size of the vesicles. These are of a globular form, and are symmetrically arranged in clusters upon an inflamed patch of skin. Each vesicle is distinct, and remains so throughout its course. It lasts about eight to twelve days, and often terminates by the formation of a thin incrustation. The eruption is attended with burning, and in the acute variety with some fever.

Herpes has seldom a longer duration than three weeks; though it may be a chronic disease. It happens usually in persons of delicate

[1] Journal of Cutaneous and Genito-Urinary Diseases, 1887.

skin; is generally limited, having its seat on the lips, eyelids, prepuce, or pudenda; and is very often associated with an internal disorder, especially with irritation of some portion of the gastro-pulmonary mucous membrane. *Herpes labialis* mostly appears at the decline or termination of fevers; sometimes at the height of acute maladies, as in pneumonia. The most distressing form of herpes is that usually extending around one-half of the trunk,—*herpes zoster*, an acute disorder, which may show itself over the course of any of the superficial nerves, and is attended by nerve-pain. Indeed, herpetic or bullous eruptions often happen over the course of the nerves, and any nerve-lesion the result of disease or of an injury will produce them. In herpes zoster around the chest, the severe pain preceding the eruption is often mistaken for pleurisy, but palpation will reveal local spots of tenderness along the course of the affected intercostal nerve.

Herpes and eczema may both be confounded with *scabies*, which, like them, occasions a vesicular eruption that is apt to be found on the inner surface of the limbs and flexures of the joints and on the dorsum of the hands between the fingers. The distinction consists in the locality affected; in the more severe itching, especially at night; in the small conical vesicles, torn, as they usually are, by scratching; and in the presence of the acarus, which may be removed from its burrow with the point of a needle or of any sharp instrument.

Bullous Diseases.—Bullæ differ from vesicles only in their size. The typical bullous disease is *pemphigus*. This affection, more common in children than in adults, appears in very large vesicles or bullæ surrounded by a slight zone of erythematous redness. The blebs occur in crops, and look like small blisters filled with serum. They are not met with on the scalp. Where there are few bullæ, we generally find them on the ankle or on the hand. The disorder may be acute or chronic. It is ordinarily chronic, and happens in persons of enfeebled constitution. Relapses are frequent, and a fatal result is common. Pemphigus may be produced by the administration of iodide of potassium,[1] or by syphilis. *Syphilitic pemphigus* is mainly met with on the soles of the feet and the palms of the hands of newly born syphilitic children. There is a form of extensive pemphigus with flaky incrustations like eczema,—*pemphigus foliaceus;* but we can still find bullæ, and there is great attending prostration. Neurotic vesicular erythema occurs after injury to a nerve, and sometimes causes blebs which may be mistaken for *herpes* or *pemphigus*, as

[1] Bumstead, Amer. Journ. Med. Sci., July, 1872.

in a case reported by Shields,[1] in which recurrent attacks of vesicular, or bullous, erythema were observed in the forearm, following the crush of a finger. The symptoms were entirely obviated by amputation of the stump of the finger, after the affection had existed for a period of three years.

Hydroa.—This is a disease like herpes, only occurring in a more diffused manner and presenting larger vesicles, arranged for the most part in the form of crescentic rings. It is a chronic condition, lasting usually from five to eight months, and there are in this period many acute or subacute outbreaks, in which the large vesicles form and then dry away. These attacks are non-febrile, and are attended with marked itching. The disorder happens chiefly in persons of depressed nervous system or gouty taint. It has been confounded with the eruption of bullæ from iodide of potassium; but these are much larger, are more persistent, and leave a marked scar. Van Harlingen considers cases of hydroa to be examples either of erythema iris or of dermatitis herpetiformis.

Pustular Diseases.—These are marked by circumscribed elevations of the cuticle which contain pus. Acne, impetigo, and ecthyma belong to this group. Rupia, too, although often classed among the bullous disorders, appertains more strictly to the pustular or to the syphilides.

Acne.—This is an eruption of hard, isolated, red elevations, due to chronic inflammation of the sebaceous follicles and the areolar tissue around them; plugs of sebum are retained in the ducts. At the apices of many of these elevations pus forms, which is discharged, leaving a hardened base, that only gradually disappears. Acne is generally seen on the face and shoulders. Men of sedentary occupations and drunkards are very liable to it. In women it is frequently associated with uterine disturbances; in men, with some digestive or genito-urinary disorder. An acne eruption also follows the use of the bromides and the iodides internally, and the local use of tar. In *acne rosacea*, lymph is generally effused into the papillary layer of the skin, and some acne pustules are seen, surrounded by the reddened, altered skin. It is a disease of years' duration, but no ulcerations happen, although scarring is a not infrequent result from the small abscesses. Unna has reported the discovery of a special bacillus in acne, but Lomry[2] is of the opinion that it is a mild variety of bacterium coli, and that the staphylococcus pyogenes albus is also present in the

[1] The Cincinnati Lancet-Clinic, May 25, 1895.

[2] Dermat. Zeitung, Bd. iii. H. 4.

pustules of acne. In ordinary *comedo*, unaccompanied by inflammation, microbes are present in abundance, the staphylococcus albus being always represented, though less numerous than in pustular acne.

Impetigo.—This is a malady often happening in persons of good general health. It presents small pustules occurring in successive crops, arranged in clusters. The pustules are isolated, are little raised above the surface, break, and a thick yellowish or greenish crust is developed; no scar follows. When the disorder attacks the scalp and face, especially in infants and children, it gives rise to extensive incrustations, and constitutes, particularly if conjoined with eczema, the affection designated as "porrigo larvalis." There is a contagious form, described by Tilbury Fox, which occurs acutely, is epidemic, preceded by fever, and unattended with pain or itching. Another form of impetigo, first mentioned by Hebra, consists in a multiform eruption of vesicles, vesico-pustules, and pustules.

Impetigo contagiosa is characterized by vesico-pustules or blebs drying into flat, straw-colored crusts. It is contagious, and is especially encountered in children. The lesions occur chiefly on the face and hands; the contents, at first serous, become sero-purulent in the process of drying.

Dermatitis herpetiformis or *Duhring's disease*, differs in being not contagious, and in its happening in older persons who are frequently of an hysterical type. Leredde and Parin, at the Hôpital St.-Louis, found in the skin, at the site of the lesions, numbers of eosinophile granules and cells. There is a close connection between this disease and the herpes of pregnancy, in which the same cellular elements have been found.[1] There is deficiency of urea in the urine of Duhring's impetigo, and Bar found that the toxicity of the urine was increased at the time that the eruption occurred. The disease is regarded by many as a cutaneous neurosis; but Leredde suggests that the exciting cause may be deficient elimination by the kidneys, and nephritis has been found by Gaston[2] in two autopsies.

Danlos[3] has reported the case of a syphilitic patient, who, after the administration of potassium iodide for a short time, suffered with a typical dermatitis herpetiformis affecting the hands, face, ears, feet, trunk, and arms. A condition, therefore, indistinguishable from Duhring's disease may occur among the rarer symptoms of iodism.

[1] Anatomie pathologique de la Dermatose de Duhring; Annales de Dermatologie et de Syphiligraphie, No. 4, April, 1895.

[2] Annales de Derm. et de Syph., Paris, April, 1895.

[3] Société Médicale des Hôpitaux de Paris, 1899.

Ecthyma.—This differs from impetigo by the larger size and greater prominence of the pustules and their inflamed base. When the crust that forms on each pustule falls, a highly-congested surface or a superficial ulceration is seen, which leaves a cicatrix. The disorder is painful, generally chronic, and connected with a cachectic state of the system; irritation of the skin may excite it. It bears a certain resemblance to sycosis; but the limitation to the hairy portions of the face, the yellow color of the pustules, their conical form and smaller size, and the brown crusts they occasion, distinguish this malady.

Rupia.—This affection produces at first bullæ, but soon large pustules, which desiccate into thick, brownish crusts, often of conical shape or resembling the shell of an oyster; when thrown off ulcerations of various depths are exposed that are slow to heal, and on which fresh crusts arise. The disease runs a chronic course. It occurs especially on the lower extremities, and is due to syphilis. It is very like ecthyma, and can be distinguished only by the history of the case, the evidences of syphilitic taint, the persistent ulcerations, and the prominent, peculiarly shaped crusts.

Squamous Diseases.—The predominant characteristic of these is the formation of small, whitish patches of unhealthy cuticle covering red papular elevations on a deep-red, dry, somewhat thickened surface; the scales are generally very freely cast off. Psoriasis is the main disorder belonging to the group.. *Pityriasis* is included by many, while others regard it as merely a variety of chronic erythema, or of eczema. It differs from lepra and psoriasis by the production of minute scales, which are constantly thrown off and reformed, and which are seated on a reddened integument; hence its chief variety is designated *pityriasis rubra*. It begins at a special point, and, unlike psoriasis, spreads over the whole body. The skin is very red, and not thickened except in instances of long standing; there is no discharge, as in eczema, nor itching or burning; the scales are loosely adherent to the surface, and at times come off in large flakes. The disease is most apparent on the body and the limbs; in chronic cases the general health deteriorates, and a fatal result is the rule. Pityriasis rubra is to be distinguished from exfoliative dermatitis, which is an acute affection and more amenable to treatment. In this disease the scales are thicker, larger, and more abundant than in pityriasis rubra; there may be some spots of moist eczema, the lesions being papular at first and then vesicular, ending in profuse exfoliation, large casts coming away from the fingers and toes. Alopecia and shedding of the nails are common.

Pityriasis rosea, or *pityriasis maculata et carcinata* of Duhring, is

recognized by the presence of maculæ, or very slightly elevated patches, varying from a pin-point to a half-dollar in size, the color being rosy, or pink, with a yellowish tint. The surface of the lesions is dry and slightly scaly; the appearance is circinate. The eruption usually appears on the trunk, is moderately acute, and may last two or three months or longer. It is not contagious, and apparently is not parasitic in origin, though this is a matter of much doubt. The general health is not impaired, and the patches give no annoyance except by the itching, which is not excessive, and by their appearance, which may lead to their being taken for lesions of syphilis, or for ringworm, lichen ruber, psoriasis, or one of the eruptive fevers, as in cases reported by Duhring and Stelwagon.

Psoriasis.—Here we find patches of a red hue raised above the surrounding integument and covered by scales of dried epidermis. The patches are infiltrated and thickened, and they often have a circular shape, with large pearly white scales. More generally the scales which completely cover the morbid portion of skin are small, though thick; the patches are large or consist of small ones which have coalesced, are not of an annular form, or completely separated by healthy skin; they are symmetrical. Psoriasis generally first appears on the extensor surfaces of the elbow- and knee-joints, and finally on the face. As Beverley Robinson has proved, the morbid change begins in the cells of the epidermis. There is no watery discharge, and scarcely any itching.

Psoriasis is often hereditary; in old persons it is frequently of gouty origin. It is a chronic affection, and extremely obstinate. It is liable to be mistaken for *lichen*, especially the isolated circular form of it, the so-called lepra. It is, however, distinguished by the distinct, dry, and silvery scales, and by the smooth, red, perhaps bleeding skin which is at once perceived when the scales are detached. Psoriasis has a predilection for the vicinity of the joints, especially the elbow- and knee-joints. Sometimes it appears exclusively on the palm of the hand; and in this form especially we are apt to find deep cracks. Palmar psoriasis is rare; but a condition resembling it in the production of scales and fissures occurs often in constitutional syphilis, the so-called *syphilitic psoriasis*. Psoriasis differs from *eczema squamosum* by the preceding vesicles, severe itching, and the want of uniformity of lesion of the latter. In scaly syphilitic eruption the scales are comparatively few and fine; when they are removed, the dense skin underneath does not bleed; and the eruption is not likely to be met with on the elbows and the knees.

Ichthyosis.—Fish-skin is also a squamous disease; but it differs

from the others of this class in involving often the whole integument, and in the absence of reddening or any signs of inflammation of the harsh, dry surface; it is, indeed, an hypertrophy of the cuticle. The skin is dry, dirty, and rough, and covered with thickened and exfoliating cuticle and with sebum; there may be also fissures and cracks. Ichthyosis is almost always of congenital origin and begins in childhood; it affects the whole body, though the face but slightly.

Among the inflammatory diseases of the skin, those resulting from medicines may be here mentioned. This *dermatitis medicamentosa* is brought about by a variety of drugs, and differs according to the special drug. Among the principal ones producing morbid appearances of the skin are arsenic, quinine, belladonna, opium, chloral, salicylic acid, antipyrin, copaiba, the bromides, and the iodides. The acneiform eruption due to the bromides, with the dusky-red color of parts of the skin, or the ulcers they may occasion; the papular or bullous eruption caused by the iodides, especially by iodide of potassium, and the scarlet rash of belladonna,—are well known.

Maculæ.—These include blood-spots, as in purpura, or spots in consequence of parasitic formations, as in tinea versicolor. But their chief cause is increased pigmentation.

First, *lentigo* may be mentioned. This consists of the little yellow or yellowish-brown spots which are so often observed on the face and on the arms in children under eight years of age, and which, if they have persisted, disappear in middle life. Similar spots are *ephelides*, or freckles; these, though aggravated by exposure to the sun, may exist all the year round. *Melasma* is a very dark pigmentation, which, although it has been met with in an epidemic form, is commonly seen in connection with Addison's disease.

Chloasma consists of a brownish or yellowish-brown pigmentation, giving rise to the so-called liver spots. They are smooth and well-defined maculæ without scales, and may result from any local irritation, or from exposure to the sun or heat. They may also happen in cases of faulty digestion with torpor of the liver, in uterine disorders, and in the pregnant state. *Tinea versicolor* is constantly confounded with these so-called liver spots. But it is almost entirely a disease of the trunk, is much more itchy, is slightly raised, and in the scales we scrape off is found the characteristic fungus.

New Growths.—These are hard, indolent, and often permanent tumors of the skin, which in their main forms consist of granulation tissue. Lupus, fibroma molluscum, and elephantiasis of the Greeks mainly illustrate this group.

Lupus.—In lupus the new growth mostly takes place in the form

of isolated tubercles. These may or may not ulcerate. They are of a dull-red color, elevated above the surface, with a well-defined outline, spread outward into normal textures, and, if they ulcerate, destroy the tissues in which they are situated. The ulcers also spread, and occasion much devastation. When they heal, they leave a strongly marked whitish cicatrix and unhealthy-looking skin. The disorder occurs in syphilitic or in scrofulous persons,—generally in the latter,—appears often in childhood, is attended with some pain and itching, and pursues a very slow course. The nose and cheek are the favorite sites. There is a form of lupus occurring in strumous subjects, and characterized by warty formations. This *lupus verrucosus* is without pain or itching, but cicatrices form, even though there have been no previous ulceration.[1] In *lupus erythematodes* the disease is superficial, and the sebaceous glands particularly are distended. The surface is somewhat raised, the centre of the diseased patch is pale and sinks in. The nodules form late, if at all, and there is no ulceration. The most common site of the disease is under the eye. It does not generally appear until after puberty, and is preceded by erythema of the affected parts. The diagnosis of *lupus vulgaris* depends principally upon the small nodules of granulation tissue deeply embedded in the corium of the skin, that have a tendency to undergo ulceration, and leave, upon healing, a peculiar cicatrix of uneven thickness. Tubercle-bacilli and giant-cells are found in these lesions, which are, indeed, due to the tubercle-bacilli.

Lepra.—Leprosy is a chronic constitutional disorder, and the symptoms of general depression may precede the characteristic local features. The true leprosy, the *elephantiasis of the Greeks*, is distinguished by tubercles, from the size of a pea to that of a walnut, of reddish or whitish or bronze-like hue, which slowly ulcerate, and which are preceded by erythematous patches; ulceration is apt to take place about the fingers and toes. Like lupus, the tubercles have the structure of granulation tissue. Often, too, there are symptoms of defective innervation, especially deficient sensation of the surface, anæsthesia of the fingers being an early symptom. The nerve-trunks are invaded, cutaneous eruptions in their course result, and the blood is seriously affected. Muscular weakness and wasting may be also present. The face is most frequently the seat of the malady, and becomes very much thickened and disfigured; similar changes may also be seen in the limbs. Pemphigus-like blebs are among the earliest signs. When marked nodules form, the skin is discolored, often

[1] McCall Anderson, Journal of Cutaneous Medicine, vol. i.

copper-colored, and the face is distorted and has a fierce expression. Sometimes anæsthesia is the main symptom; the uneven thickening may occur, without tubercles, in circular patches like psoriasis, and be markedly anæsthetic. The disease is often hereditary. Syringomyelia, presenting trophic changes, closely resembles anæsthetic leprosy; and Zambaco[1] has shown that cases supposed to be typical illustrations of Morvan's disease and reported as such were in reality cases of leprosy. The presence of the lepra bacillus in the lesions or in the blood would decide the diagnosis in any doubtful case.

Two forms of the disease are recognized,—the tubercular and the anæsthetic; but there is no absolute distinction between them. The disease is found in the east, in Africa, in Brazil, in Norway, and in the Hawaiian Islands; a few cases exist in the United States.

Hypertrophies.—There are many forms of these, according to whether the connective tissue, the epidermis, the arteries and veins, or the lymphatic vessels are affected. I shall notice particularly two; and first, elephantiasis Arabum.

Elephantiasis of the Arabs.—This, the Barbadoes leg, is an enormous increase in size of the limb, usually dependent upon an indurated swelling of the subcutaneous tissues, with some alteration of the skin proper, and lymphangitis. The tumefaction may be in swellings separated by deep furrows, giving somewhat of a tuberculated look to the part, or it may be uniform; it chiefly attacks males, and occasions great deformities. It is a disease of the tropics. Cases, especially of elephantiasis of the scrotum, have been frequently traced to filariæ, or to repeated attacks of erysipelas.

There is a form of enlargement of the leg to which we may here briefly refer,—one in which the overgrowth of the affected limb is associated with *disease in the lymphatic system*. Vesicles form, which are connected by ridge-like elevations, and which from time to time discharge a chylous fluid.[2] The subcutaneous lymphatics near the groin are usually found to be distended.

Scleroderma.—Scleroderma, or sclerema, is an induration of the skin and areolar texture, which may be partial or general, affecting nearly the whole body. The skin is dense and hard, and in the true skin and the subcutaneous tissue the fibrous elements are much increased. The true skin shrinks and binds down and is bound to the parts beneath. If the malady seize upon the fingers, it renders them rigid. The disease is generally symmetrical, and much more common

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[1] See Gould's Year-Book of Medicine and Surgery, 1897, p. 860.

[2] W. H. Day, Transact. Clin. Soc. Lond., vol. ii., 1869.

in women than in men; it may appear after unusual exposure to cold and resulting frost-bite.[1] It frequently coexists with feeble health; and in time the internal organs become affected, or these are from the first implicated.[2] Yet the general health may remain good. In generalized scleroderma the plaques may appear on any portion and extend over almost the whole surface of the body, as in the case described by Leredde and Thomas.[3]

I had some years since a marked case of this strange affection under my charge at the Pennsylvania Hospital, in a woman, forty-two years of age, who, admitted with œdema of the feet, was at the same time noticed to have a swelling of both wrists and forearms as well as of the cheeks. The swelling was firm and resistant, and did not pit on pressure. The skin covering it was very smooth, and of redder hue than at other portions of the body; there was well-preserved sensibility. The œdema disappeared from the feet, but the signs of the indurated cellular tissue did not leave the affected parts. On the contrary, the condition of these parts became worse, though the general health was excellent, all the internal viscera being in a normal state. Gradually the hands, particularly the fingers, were found to be more and more resisting and immovable, and she could scarcely bend them; occasionally they were the seat of pain. The skin lost all suppleness, and could not be raised up. At no time while under observation was albumin present in the urine. She left the hospital unimproved by the sulphur baths, the bichloride of mercury, and the various other alteratives she took; and I afterwards learned that she died of an acute pleurisy succeeding an attack of acute meningitis. Prior to her death, so great was the pressure exerted by the dense and contracting areolar tissue that dry gangrene of a finger ensued, as well as of a toe, the disease having been also noticed in the lower extremities. She died about one year from the beginning of the disease. Examined after death, the skin of the affected parts was found firmly united to the muscles by the dense areolar textures.

Scleroderma is very similar in many of its features to *myxœdema*. But the marked anæmia of this, the decided nervous symptoms, and the fact that we do not find the stiff, hard skin compressing the parts beneath causing in time marked atrophies, distinguish the two maladies. The successful treatment of *myxœdema* by thyroid extract suggests a means of diagnosis between the two affections. Goldzieher[4]

[1] Goldzieher, Beiträge der Berlin. Dermatolog. Gesellsch., March, 1893.

[2] Harley, Med.-Chir. Transact., 1877.

[3] Archives de Méd. Exp. et d'Anat. Path., Sept. 1898.

[4] Beiträge der Berlin. Dermatolog. Gesellsch., March 12, 1893.

considers scleroderma a progressive chronic dermatitis accompanied by permanent œdema. Repeated attacks of *erysipelas* thicken the skin, but we do not find atrophies from compression.

Scleroderma is closely related to *morphœa*. This occurs over the course of nerve-tracts, the thickening being in circumscribed patches and lacking the peculiar hardness of sclerema; then changes in the structure of the skin, hyperæmic appearances at first, pigmentation and cicatrization afterwards, occur in morphœa, with pain and tingling in the affected parts. The color of the patch of morphœa is characteristic. The central part is usually of a yellowish-white or ivory color, which is surounded by a zone of lilac, due to enlarged capillaries. By some, morphœa is regarded as a local expression and an early stage of scleroderma, each being a form of trophoneurosis. There is a possibility of mistaking the *local asphyxias* occurring in symmetrical patches and characteristic of Raynaud's disease for scleroderma, but the course of this disease is short, lasting only ten days, and ending either in gangrene or complete restoration of function. It should be remembered, however, that Raynaud's disease is not confined to the digits, but may occur on the forearm or leg, and other portions of the body. The local asphyxia of the fingers met with in hysterical patients is a fugacious phenomenon, and is not caused by any organic change in the nerve-supply. It usually takes place at night and passes away in the morning.

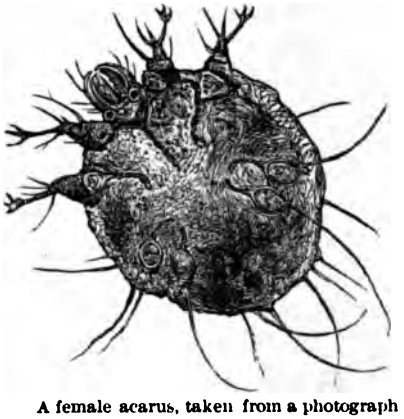
Parasitic Diseases.—These may be caused by the presence either of parasitic animals or of parasitic plants. To affections of the former origin, or the epizoa, belongs especially scabies; though the various forms of lice producing a pruriginous eruption with little hemorrhagic marks—phtheiriasis—must be mentioned. Another animal parasite, the entozoon or demodex folliculorum, inhabits the sebaceous and hair-follicles, but does not cause disease.

The complaints associated with the vegetable parasites, the *epiphytes*,—or, as those on the skin are called, the *dermatophytes*,—also known by the generic name of *tinea*, are chiefly favus, mentagra, pityriasis versicolor, and some of the forms of ringworm, *tinea circinata*, and *tinea tonsurans*. *Pellagra*, supposed, too, to be due to a vegetable parasitic growth, is not an affection met with in this country. Nor does the presumed parasitic fungus lodge in the skin. It is said to be found in diseased Indian corn or maize, which, when eaten, causes the digestive disorders, the general cachexia, and the erythematous cutaneous eruption that characterize the malady. In the chronic cases melancholia, suicidal mania, and paraplegia are met with. Belmondo found lesions of the spinal cord in a number of instances.

Scabies.—Scabies, or the itch, is owing to the acarus scabiei. This burrows in the skin, particularly between the fingers and between the toes, about the wrists, on the buttocks and abdomen, and the upper part of the penis. The channels produced are curved, and may be traced as whitish or more generally black streaks several lines in length, in the situations just indicated. The disease is attended with excessive itching, which is increased at night, and with the eruption of conicle vesicles, or even of a marked eczema and of papules and pustules; most of the rash is due to the irritation of scratching.

At the close of our civil war we had a form of itch prevalent in this country, spread far and wide, as is presumed, by contact with the troops,—the so-called *army itch*. It was a chronic and distressing affection, and no age or social state was exempt from it. The itching was intense; the eruption was like prurigo, but vesicles, or even an eczematous condition of the skin, or pustules, attended the intolerable itching; and in cases of long duration the appearance of the skin was altered, and all trace of a distinctive eruption was gone. The eruption was seen on the arms, chest, abdomen, and lower extremities, particularly on the ulnar side of the forearm and the inner aspect of the thigh. It was sometimes found on the scalp, but seldom in the groins, in the axillæ, on the hands, or between the fingers. It was benefited by sulphur; for almost all the preparations recommended for it contained sulphur. To what it was due I am unable to say.

FIG. 86.



A female acarus, taken from a photograph from nature: magnified 220 diameters. The ventral surface is shown.

Tinea Favosa.—Tinea favosa, or favus, is a chronic disease that gives rise to bright-yellow umbilicated crusts, of circular shape, which often form yellow rings around the hair-follicles. There is no discharge. The disease rarely affects any other part of the body than the scalp, and produces baldness; when the nails are attacked, they become brittle and yellow. The microscope furnishes us with a certain means of diagnosis, by exhibiting the cryptogamic plants.

Tinea Sycosis.—Tinea sycosis, or barber's itch, is to be distinguished from a non-parasitic form. The distinctive marks of the disease consist in the development of yellowish pustules, having a

bright-red base, around the roots of the hair of the beard: the hairy portion of the neck may be also affected. The crusts may run together, and more or less inflammatory thickening of the skin exist. This is especially seen in the parasitic form of the disease, in which, however, less suppuration happens, and less pain or itching, but in which the hairs become brittle. The upper lip is rarely implicated in tinea sycosis. Non-parasitic sycosis consists chiefly in an inflammation around the follicles, which always starts in these parts.[1]

Tinea Circinata and Tinea Tonsurans.—The tricophyton tonsurans is the parasite met with in *tinea circinata*, the ringworm of the body, and in *tinea tonsurans*, the ringworm of the scalp. This is common in children, and spreads by contagion. It exists in circular scaly patches, on which are dry broken hairs. In ringworm of the body the patches are also circular and scaly: but they are red and very itchy, and much paler in the centre than at the edge. Examining the scurf, we find the fungous growth.

Tinea Versicolor.—This parasitic affection, also known as pityriasis versicolor, occasions those yellow or yellowish-brown discolorations which may be not infrequently seen on various parts of the body. The microsporon furfur of Eichstädt is the parasite present: and it is found abundantly in the scales which can be scraped from the raised, itching patches. In pityriasis affecting the scalp we may also find parasitic growths of a vegetable nature: they are often the cause of baldness, as in *tinea decalvans*. Pityriasis capitis, or dandruff, is readily distinguished from ordinary seborrhœa, in which the oily element predominates in the scales, that are aggregated in masses. This is in marked contrast to the fine pearly scales of pityriasis, which are due to epithelial exfoliation. This condition often leads also to baldness.

The diagnosis of *actinomycosis* of the skin depends upon the history, and the distribution of the tumors in necklace-like series, either in lines or in circles, or in groups. The disease pursues a rapid course, with fever, sometimes septicæmic in character. Majocci[2] recognizes two forms, the anthracoid and the ulcero-fungous. In the former the lesions are flat-topped, with a multitude of small openings from which thick yellow pus exudes: in the latter ulceration occurs early, with large granulations. In neither form are the lymphatic glands in the neighborhood of the lesions involved. The lesions are situated chiefly around the buccal cavity. Microscopical examination of the yellow granules reveals the characteristic actinomyces.

[1] Robinson, New York Medical Journal, Aug. and Sept. 1877.

[2] Annales de Derm. et de Syph., Paris, 1892, p. 310.

Altered Gland-Secretions.—One of these, *seborrhœa*, or increased secretion from the sebaceous glands mixed with epidermic scales, has been already mentioned. It is especially found on the scalp, nose, and genitals, and is often seen among those who have menstrual disorders. It is unattended by itching; the crusts are readily removed by strong alkaline soaps, and the skin beneath is healthy, or pale and glistening, or slightly reddened. Where the sebum is retained in the follicle, giving rise to little prominences discolored by dirt, and without, as happens in acne, inflammation around the gland and its duct, the disorder is called *comedo*. The plug of sebum can be easily squeezed out. The disorder is most common on the face and shoulders of young persons of lymphatic temperament.

The *sweat-glands* are often altered in their activity, and excessive perspiration results. This may be general, or confined to particular localities, as to the hands and feet. This local sweating is often offensive, and makes the parts very tender. The disease formerly known as *lichen tropicus* is now regarded as due to congestion or inflammation of the sweat-glands, and is termed more correctly *miliaria papulosa*. The strophulus or "red gum" of infants is *miliaria vesiculosa*. At times there is sweating of blood from the skin. This condition, known as *hæmidrosis*, is due to some alteration in innervation, and may be preceded or accompanied by a localized erythema or eczema; or the bleeding may come from the follicles of the skin; it is not a secretion of the sweat-glands, but is a hemorrhage, or an exudation.

Molluscum epitheliale presents numerous globular or flattish nodules, sometimes seated on a broad base or attached to a pedicle. They are due to a psorosperm in the deep layers of the skin and in the sebaceous glands. The lesions occur in groups on the face or neck, or on the trunk; they have a doughy feel, vary in size from that of a pea to that of a pigeon's egg, show no tendency to inflame, and are not attended with increased sensibility of surface. They are of the color of the skin or of brownish hue. They may last during life without affecting the general health. There is a variety met with especially in children, which has at the top or the side of each tubercle a small orifice, from which a creamy, fatty fluid can be pressed. This variety is by many regarded as contagious. The little tumors are distinguished from so-called *molluscum fibroma* by the central aperture, and by the substance resembling sebum that can be squeezed out of them.

Although *plica polonica* is of rare occurrence in this country, yet among immigrants it is occasionally met with, and may be recognized by the mass of felted, matted hair, and the inflammation of the scalp from which serous oozing occurs. The mass of hair affords refuge for

vermin, and the secretions from the scalp produce a peculiar odor, which has led to the supposition that the disease is caused solely by dirt. Dumesnil considers it a neurosis and the dirt only incidental. Under the microscope the hairs show decided change, affecting mainly the medulla. Jarochevski has pronounced the disease a disturbance of nutrition of neurotic origin.

Nervous Affections.—Several of these, such as herpes zoster, have been already considered. The large group of itching affections in which no obvious local affection exists, find here their place. Such are, for instance, the various forms of *pruritus*, either local or general, which are specially apt to affect elderly persons. Sometimes the itching is violent and obstinate, and we cannot even trace it to reflected irritation, though this is often its cause. Again, diabetes, gout, lithæmia, or jaundice may lie at the root of the pruritus. Season influences it much, as seen in the winter itching, the pruritus hiemalis, described by Duhring. It happens particularly about the thighs and legs, and there may be prominence of the hair-follicles. Among other manifestations of nervous skin disorders are dermatalgia, hyperæsthesia, and anæsthesia; then there are a number of cutaneous diseases that are being recognized as of nervous origin.

The affections of the skin do not always occur isolated: they may be combined. Again, they are altered by the existence of a special taint, as by the syphilitic. Now, without attempting to describe *syphilitic* diseases of the skin, it may be stated that they differ by their multiform lesions, their copper-colored tint, by the stained aspect they leave, and by the absence of itching. In syphilitic erythema the eruption runs a chronic course, and is distinct on the trunk. It belongs to early syphilis. Syphilitic lichen has better-defined papules than simple lichen. The ulcerations in the pustular affections are deeper: while in the squamous disorders the scabs are smaller and the papules larger than in the non-syphilitic eruptions. A furunculoid eruption is met with in hereditary syphilis. Syphilitic affections of the skin are apt to be mixed, and light is thrown on them by this fact, as well as by the history of the case, the sore throat, the falling of the hair, and the nerve- and bone-pains.

CHAPTER XIV.

POISONS AND PARASITES.

Toxic symptoms from causes arising within the body, either from fermentative or putrefactive changes of the food within the intestinal tract, or from micro-organisms causing infectious diseases, septicæmia, sapræmia, and the like, have been referred to in other chapters, especially those on Diseases of the Blood, the Acute Infectious Diseases, and Gastro-Intestinal Affections and Fevers. In this section will be considered only those disorders due to poisons or parasites, the morbid phenomena of which are clearly occasioned by causes introduced into the system from without.

POISONS.

Cases of poisoning may arise from accident, attempt at suicide, or criminal intent. It is only intended here to set forth the main signs by which the most common poisons may be recognized and distinguished. For this purpose it will be convenient to consider the cases as divided into acute and chronic, subdividing these classes according to the character and effects of the different substances.

Acute Poisoning.

The attack comes on suddenly, the patient, previously in good health, having taken some food, drink, or medicine which has been followed by the severe symptoms. It is always, in a case of suspected poisoning, of the utmost importance to be able to make out these points.

Irritant Poisons.—The chief articles which give rise to acute poisoning belong to the class of irritant poisons. The symptoms are generally those of acute gastritis, attended often with more or less inflammation of the mouth, the fauces, and the œsophagus. Sometimes the air-passages may be involved, either directly or by sympathy, and we find hoarseness and cough. Convulsions are occasionally observed, and collapse is apt to occur sooner or later.

The acute pain, the tenderness, and the vomiting come on shortly after a meal, or at least after something has been swallowed. This

distinguishes the acute gastritis caused by poisons from *idiopathic acute gastritis* or from *acute gastric catarrh*. Sometimes several persons are similarly affected,—a circumstance always strongly in favor of the idea of poisoning. From *perforation of the stomach* or *intestines*, irritant poisoning is discriminated by noting that the acute signs in the former case follow upon the manifestations of some gastric or intestinal disorder; and the attending phenomena of collapse are not, as in poisoning, associated with cramps or convulsions. *Cholera morbus* is separated by the history of the case, by the absence of epigastric tenderness, and by the purging and vomiting often coming on simultaneously. *Cholera* resembles poisoning in the suddenness and the violence of the attack, but is distinguished by the rice-water discharges and by its epidemic character. Bacteriological examination of the stools also affords a means of diagnosis. In *strangulated hernia*, the comparatively gradual onset, the pain, the tumor, and the constipation will be significant. As regards the separation of cases of poisoning in which blood is ejected, from ordinary *hemorrhage from the stomach*, we find that pain and purging are both absent in the latter, while in irritant poisoning they are well-marked symptoms.

Let us now examine some special poisons. Strong acids are sometimes used in self-destruction. *Nitric* acid colors the lips and mouth orange-yellow wherever it touches them. *Sulphuric* acid stains the skin or mucous membrane white or even dark gray; the pain is excessive, and nervous symptoms are not infrequent. If the vomited matter be mixed with a solution of barium nitrate, a dense white precipitate of barium sulphate is thrown down. *Hydrochloric* acid is less irritant and corrosive than sulphuric acid; in the ejected matter silver nitrate produces a copious white precipitate insoluble in nitric acid. *Oxalic* acid, when concentrated, is rapidly fatal. The irritant effects are those of the mineral acids; but we also meet with dyspnœa and with nervous phenomena, such as anæsthesia, paræsthesia, palsies, and convulsions.

The strong *alkalies*, when taken into the stomach, cause inflammation of the organ and of the fauces and the œsophagus. Should the case end in recovery, thickening of the œsophagus is apt to occur. *Ammonia* may also induce severe nervous symptoms, similar to those of tetanus; its vapor sometimes acts powerfully on the air-passages, producing harassing cough. *Potassium* and *sodium hydroxides*—commonly known as caustic potash and caustic soda—give rise to violent local inflammation in the mouth, œsophagus, and stomach. The vomited matter has an alkaline reaction. *Potassium nitrate* is a strong cardiac sedative.

Potassium iodide, *iodine*, *bromine*, and *chlorine* are all capable of destroying life by their intensely irritant effect.

Phosphorus, which is not infrequently taken as a poison, imparts to the breath, to the fæces, and even to the urine an alliaceous smell, and may make them luminous in the dark. It acts as an irritant, causing obstinate vomiting and purging, pain at the epigastrium, rapid, weak pulse, jaundice, and unquenchable thirst. The local pain and inflammation are usually extreme, and collapse, with or without convulsions, comes on early. In some cases painful cramps in the limbs occur, and various disturbances of sensibility, and, later, violent delirium and convulsions, eventuating in coma and in death. In other cases hemorrhage is a striking feature, the blood is very fluid, and issues from all the passages, and petechiæ form beneath the skin. The temperature remains normal until near death. The pulse becomes feeble and small; the first sound of the heart almost disappears. Jaundice is a constant symptom; it seldom, however, comes on before the third day, and is rarely intense; it may be associated with urticaria. The spleen increases in size simultaneously with the liver. The urine becomes very scanty. Albumin, blood, and casts are occasionally present; biliary coloring-matter is usually met with; urea is defective; peptonuria is observed. In cases of phosphorus poisoning, acute and extreme fatty degeneration of the tissues happens. It occurs with astonishing rapidity. It has been seen, in the bodies of persons poisoned by phosphorus, within forty-eight hours, and has been found to affect the heart, the smaller blood-vessels and capillaries, the liver, the kidneys, the glands of the stomach, and the voluntary muscles;[1] the liver is principally implicated.

Various compounds of *potassium*, *copper*, *zinc*, *silver*, *lead*, and *iron* occasionally cause death. They act, for the most part, as irritants merely; but some of them are powerfully astringent, and even caustic, as, for instance, zinc chloride or silver nitrate. If the toxical phenomena are due to the nitrate of silver, the staining of the lips may afford a clue to the nature of the case. There are no really distinctive symptoms produced by large doses of *arsenic*, of *antimony*, of *mercury*, or of their compounds, which are among the best known of irritant poisons: the peculiar effects of each of these substances, when insidiously introduced into the economy, will be presently mentioned. In *acute arsenical poisoning*, besides the pain and the gastro-enteric symptoms, convulsions, delirium, palsies, and bloody or albuminous urine have been specially noticed. Arsenical poisoning is a very com-

[1] Tardieu, Étude médico-légale sur l'Empoisonnement, 1867, p. 445.

mon form of self-destruction. It is also observed among those who accidentally take Scheele's green, or among children who swallow arsenical paints. There is in the internal organs a fatty degeneration similar to that in phosphorus poisoning. In the recognition of the cause of the symptoms, Reinsch's test, applied to the vomited matter, is very convenient and satisfactory. In poisoning by *corrosive sublimate*, epigastric pain, vomiting, diarrhœa, bloody stools, and finally collapse, are met with.

Among animal substances, *cantharides* has sometimes been productive of poisonous effects; strangury, bloody urine, albuminuria, more permanent than that produced by turpentine, priapism, and spasm of the glottis, are the most marked symptoms; while the shining green particles of the drug, if taken in substance, have been detected in the vomited matters.

Sausage, *milk*, *cheese*, *eggs*, especially in articles of confectionery, such as *cream puffs*, frequently produce violent symptoms suggesting some of the more powerful irritants, although chemical examination fails to reveal any mineral poison. The main cause of these actions is that under the influence of certain micro-organisms the albuminous matters undergo rapid decomposition, producing nitrogenous bodies, among which one has been identified as a *diazobenzene* compound. Vaughan originally called this body tyrotoxicon,—cheese poison. It is highly poisonous, but also very unstable. It is produced early in the decay of the albuminous articles, and is decomposed subsequently. We can, therefore, understand why articles of food may be less irritating, when decidedly decomposed than when decomposition has just set in. Besides the signs of gastro-intestinal irritation, vertigo, headache, marked anxiety, and muscular weakness have been noticed among the effects of these ptomaines.

The vegetable irritants are mainly articles commonly used as purgatives. Thus, *elaterium*, *aloes*, *colocynth*, and *colchicum* have all proved fatal when taken too freely. The symptoms do not differ materially from those caused by other poisons of this class. *Tobacco* and *lobelia* are powerful local excitants, occasioning emesis and purging, with a speedy collapse of the system. The former, when the nicotine produces acute symptoms of poisoning, gives rise also to salivation, cold sweats, slow pulse, colicky pains, and at times convulsions. *Savin* not only produces inflammation of the alimentary canal, but is apt also to give rise to strangury; it is most frequently resorted to with the view of bringing on abortion. *Ergot* is also used for the same purpose; the most striking symptoms of acute ergot poisoning are colic, vomiting, diarrhœa, increased salivation, retardation and

weakening of pulse, muscular weakness, and, in severe instances, stupor. The poisoning rarely ends fatally.

Poisonous fungi, such as the fly fungus, which are eaten by mistake for mushrooms, produce violent symptoms of irritant poisoning attended with other phenomena. The poisonous agent in the fly fungus is *muscarine*, and it gives rise to vomiting, violent colic, and diarrhœa, besides slowing of the pulse and the breathing, and violent excitement followed by stupor and somnolency. The case generally lasts two or three days, and may then end in recovery or in collapse; but it may terminate fatally in six or seven hours, hæmoglobinuria being among the symptoms. Finding the fungi in the vomited matter or in the stools greatly facilitates the diagnosis. Other poisonous fungi produce much the same symptoms; and even the usually eaten and innocuous kinds of mushrooms may, if at all spoiled, or in certain individuals, or when eaten raw, occasion similar symptoms.

Narcotic Poisoning.—The symptoms of narcotic poisoning vary more, according to the special article taken, than those caused by irritants. Narcotic poisons affect chiefly the nervous system and the circulation. Many of them produce phenomena like apoplexy and intoxication, from which they need to be carefully distinguished. Narcotic poisoning is, for the most part, of the acute form.

Opium is by far the most important of narcotic poisons. It induces giddiness, stupor, and lethargic sleep, from which, however, the patient can at first be roused, if sharply spoken to. Subsequently this sleep deepens into coma and cannot be broken; the skin is relaxed and perspiring; the face is usually pale; the pupils are contracted and insensible to light; erections of the penis are common. A more or less evident odor of opium may often be perceived about the person or on the breath. No distinction can be drawn between the effects of different forms of this poison: the stronger the preparation, however, the more marked and the more rapid will be the progress of the case. Morphine, codeine, narcotine, and the other alkaloids give rise to similar symptoms, but the smell of opium is absent; convulsions are most likely to occur from narcotine, papaverine, and thebaine.

The diagnosis of opium poisoning from *apoplexy* and from the coma of *uræmia* has been discussed in a former chapter. We may merely recall that the contracted pupil caused by opium is of very great significance, and does not, with the exceptions there referred to, exist in the other states. Moreover, the coma in apoplexy is at once developed; while in narcotic poisoning it is not sudden, but is preceded by drowsiness or stupor, which gradually passes into coma.

These phenomena occur also in the same sequence in uræmia; but they are even slower in their progress, and are frequently associated with convulsions and with markedly albuminous urine and dropsy.

From *acute alcoholism* we discriminate opium poisoning chiefly by the absence of the alcoholic odor, the slow respiration, and the presence of morphine in the urine. The characteristic smell of *chloroform*, the great pallor of the countenance, the complete and speedy collapse, and the absence of contracted pupils distinguish chloroform poisoning from opium poisoning. It is the same with *ether*. Poisoning by chloroform or by ether is mostly encountered during surgical operations.

Chloral, in excessive doses, produces heavy sleep, with contracted pupils, but they dilate on awaking.[1] There is some reduction of temperature, with rapid pulse, giddiness, inability to walk straight, double vision, and headache, in cases in which consciousness, sensibility, and muscular power have not been entirely suspended by the drug. Weak action of the heart is another of the dangers of chloral poisoning, and I have known a dilated heart almost paralyzed even by small doses. In some instances a stage of excitement like alcoholic intoxication precedes the narcotism. The urine may or may not contain sugar.[2] Chloral itself simulates sugar in the results of the copper and bismuth tests. It is occasionally used for drugging liquor for purposes of robbery or rape.

Benzin, when taken internally, occasions noises in the head, muscular tremor and twitchings, and deep sleep; but the narcotic depression ends in recovery.

Alcohol, if taken in large quantities and not much diluted, gives rise to symptoms like those caused by opium. The eye is injected and the seat of ecchymosis; the pupils are, as a rule, dilated and very sluggish; the breathing is irregular and stertorous; the temperature lowered; the insensibility may alternate with convulsions; the breath has a strong smell of alcohol or may be quite free from spirituous odor. This absence of odor of the breath, although not usual, may give rise to a confusion between alcoholic poisoning and apoplexy, and the discrimination of these conditions must then depend in some measure upon evidence furnished by the history of the occurrence of the insensibility, and by the presence or absence of palsy.

Alcohol may readily be detected in the urine. Woodbury's[3] mod-

[1] Taylor on Poisons, 3d edit., 1875.

[2] See a case of mine recorded in a Clinical Lecture on Chloral Poisoning, Phila. Med. Times, March, 1883.

[3] Philadelphia Medical Times, March, 1883.

ification of Ainstie's test is very convenient. Into a tube containing a gramme of sulphuric acid, which should be colorless or nearly so, twice as much of the urine to be tested is poured. A small crystal of bichromate of potassium is then dropped in, and the liquid slowly mixed by rotating the test-tube. If alcohol be present in proportion as large as two or three parts per thousand, a permanent green discoloration will result; if there be less than this, the liquid will remain of ruby color. Chloral in the urine does not produce the peculiar reaction.

Belladonna, or its active principle, *atropine*, and *hyoscyamus* produce more marked excitement of the brain than opium does, causing delirium of active kind, perhaps with convulsions. The pupils are greatly dilated, and vision is singularly deranged; there is intense thirst, with great dryness, redness, spasm, and burning in the throat; the breathing is rapid, thus differing from apoplectic conditions. The temperature is always lowered; the pulse becomes quick and compressible; a scarlet efflorescence may happen. The surest test of poisoning by atropine is to take some of the urine passed, and with it to dilate the pupil in the eye of a cat.

Conium occasions stupor, paralyzes the muscular system, and dilates the pupils; there is dyspnœa, while the heart, though rendered slower, is not much affected. Convulsions may come on. These help to distinguish conium poisoning from curare poisoning, which it much resembles. In the latter, however, the palsy is greater.

Carbolic acid, if taken in poisonous doses, produces rapidly dangerous symptoms, which may terminate in death in a few hours. Vomiting, slow pulse, noisy breathing, loss of consciousness, deepening into profound coma, abolition of reflex movements, cool skin, suppression of urine, are the main symptoms. When the urine is obtained, it is of dark-green or black color; this and the odor of carbolic acid about the patient are significant features. The discolored urine contains blood-corpuscles, epithelium, and tube-casts.

Aniline poisoning is met with among the workers in factories in which the aniline colors are made. It is the breathing of the aniline vapor, especially, which occasions the toxic effect. Vertigo, headache, a sense of suffocation, vomiting, anæsthesia, pain in the extremities, somnolency, and a dark cyanotic discoloration of the ears, the nails, and the mucous membrane of the nose, have been especially noticed.

Hydrocyanic or *prussic acid* usually leads to convulsive contractions of the muscles of the limbs and trunk, and destroys life by stopping the circulation and the respiration. Sometimes the odor of the acid, resembling that of bitter almonds, is perceptible in the

breath; but too much reliance must not be placed upon this point. Unfortunately, the diagnosis of this poison has generally to be made after death, for medico-legal purposes.

The gases arising from burning *coal* and *charcoal* may cause death by asphyxia; and a knowledge of this fact has, particularly in France, led to many suicides. In those cases in which the asphyxia has not a fatal termination, yet has been decided, disorders in the peripheral nerves may manifest themselves, either by the signs of neuritis, or by pain and swelling simulating a phlegmon, or by vesicular eruptions in the course of an affected nerve. The peripheral disturbances may appear at once or not until after some days. The signs of disorder of the vasomotor nerves do not last long: those of the motor or sensitive nerves have a longer duration: the complaint induced may be incurable, extending from the centre to the periphery, or in the reverse direction; or, lastly, the affection may cause an acute ascending paralysis.[1]

The poisonous action in these cases is due largely to carbonic oxide, carbon monoxide, a gas which has a strong affinity for hæmoglobin, and suspends the oxygen-absorbing function of the blood, thus establishing a chemical asphyxia. The gas, being non-irritating, may be inhaled without exciting immediate suspicion. The so-called water-gas contains large amounts of carbon monoxide. Experiment has shown that such gas is much more dangerous when inhaled than the ordinary illuminating gas, which consists almost entirely of compounds of carbon and hydrogen. In poisoning by carbonic acid, carbon dioxide, there is much greater disturbance of breathing than in carbonic oxide poisoning.

Antipyrin given in large doses may produce extreme lowering of the temperature and collapse. Cyanosis, frequency of respiration and of pulse, dyspnœa, a feeling of extreme heat over the body, and an erythematous, urticarial, or measly eruption, have also been noticed. In one instance reported, the use of the drug led to the formation of membranes in the mouth, and to symptoms of laryngeal spasm, which was not the case when phenacetin, antifebrin, or exalgin was substituted.[2]

Petroleum taken in excessive quantities produces giddiness, faintness, and palpitation, with tonic and clonic convulsions, contracted pupils, hot skin, and slow pulse; it does not occasion either stupor or vomiting: the urine has an aromatic odor. Recovery is the rule.

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[1] L[illegible], Arch. Gén. de Méd., May, 1865.

[2] Schmeer, Amer. J. Pharm. Med. Sc., May, 1890.

Nitroglycerin occasions a throbbing headache increased by motion, mental confusion, flushing of the face, pulsations all over the body, arterial relaxation, and collapse.

Following these poisons, which are in the main narcotic poisons or belong to the group of poisonous carbon compounds, we shall examine some forms of acute poisoning produced by certain powerful vegetable poisons.

Aconite has a strongly sedative influence upon the action of the heart, brain, and spinal cord, as well as an irritant action upon the alimentary canal; slow pulse, giddiness, delirium, numbness, and tingling of the skin, loss of power in the legs, with formication, tingling of the tongue, vomiting, and purging, are followed by syncope and death.

Digitalis causes dilatation of the pupil, generally with vomiting, often with purging and with headache, giddiness, and suppression of urine; its chief effect, however, is upon the pulse, which is strikingly lessened both in frequency and in force, and becomes irregular; the action of the heart, too, becomes weak, and blood-pressure is diminished. The skin is cold, pale, and covered with sweat; the mind is generally clear, though there are great lassitude, with muscular debility, a tendency to sleep, and at times convulsions. The action of the poison generally extends over days. *Veratrum viride* resembles digitalis in its action. It markedly reduces the pulse, and gives rise to vomiting, to great prostration, and to irregular breathing. The temperature is much lowered. Poisoning by *jaborandi* or *pilocarpine* produces profuse sweating and salivation, vomiting, diarrhœa, respiratory and cardiac distress, dimness of vision, and contracted pupils.

Calabar bean acts as a direct sedative to the spinal marrow, particularly to the medulla, and produces great muscular debility or relaxation, or even paralysis, extending to the heart and respiratory muscles. The mental faculties remain unaffected, and in this its action differs from that of the cerebral sedatives. It is, however, irritant to the alimentary canal, causing vomiting or purging, a peculiar epigastric sensation is generally experienced, and increased salivation is met with. Calabar bean contracts the pupil and also the ciliary muscle, thus making the eye myopic. The condition of the eye is the main diagnostic sign that distinguishes poisoning by calabar bean from poisoning by curare or by conium.

Strychnine and *brucine*, the active principles of nux vomica and of allied plants, give rise to phenomena strongly resembling those of *tetanus*. A very short time, however,—from a few minutes to an hour or two,—will determine the issue of a case of poisoning; while teta-

nus may run a course of several weeks. The first symptom of strychnine poisoning is a sense of suffocation and dyspnœa, followed by spasms of the respiratory muscles, by starting and twitching and rigidity of the arms and legs, especially of the extensor muscles, but not by lock-jaw; tetanus, on the other hand, comes on with setting or locking of the jaws, and the limbs are not at first affected with spasms; indeed, the arms remain throughout nearly free from them, and the paroxysms of spasm do not follow one another so rapidly as in strychnine poisoning, and are of shorter duration. Again, idiopathic tetanus is extremely rare: almost always there has been some wound or injury as a proximate cause of the malady. But we need not pursue these points of diagnosis farther: they have been mentioned in connection with tetanus. From *epilepsy* strychnine poisoning differs by the unimpaired consciousness; from *hydrophobia*, by the absence of spasm of the œsophagus and of the terrible dysphagia.

Picrotoxin also produces convulsions which may be mistaken for those caused by strychnine. But they are not of a reflex nature, and reflex spasms are not induced. The breathing is rapid: the contraction of the heart is retarded: there are often somnolence and muscular debility. A scarlatinal eruption has been noticed.

Chronic Poisoning.

When the patient has been subjected to the continuous action of a noxious substance, the case is said to be one of chronic or slow poisoning. Any of the irritant poisons, given in small and repeated doses will keep up a morbid condition of the stomach and bowels much like ordinary chronic inflammation.

The narcotics, taken in the same manner, act upon the vasomotor nerves and the cerebro-spinal system, and through this upon the alimentary canal, so deranging digestion and nutrition as even indirectly to cause death. *Opium* is the most important of the articles thus used: it is often administered to infants for the purpose of quieting their cries, and the frequent repetition of the dose induces a series of phenomena closely allied to those observed in the adult. With the effects, on the mind, of opium taken persistently for the sake of intoxication, the reading world is familiar through the published experiences of De Quincey and of Coleridge.

The habit is here and in Europe generally acquired only by persons who have begun the practice for the relief of some painful affection: in the East, opium is used much more commonly, and, in many Oriental countries, to smoke it is a favorite amusement. Those who employ it constantly are pale, or have a sallow, haggard countenance and

a dull eye. They lose their power of will and their energy, and are troubled by loss of appetite, giddiness, anomalous neuralgic pains, sleeplessness, and low spirits, which they relieve by resorting to the opiate. Though, in spite of the pernicious custom, the general health may remain for many years good, yet sooner or later it gives way, and the opium-eater dies worn out; or death may be the consequence of disease of the liver, of palsy, or of inveterate diarrhœa, produced by long addiction to the vice. Persons who consume large quantities of opium are apt to have, from time to time, attacks of extreme nervous prostration, attended, perhaps, with violent headache, and requiring free stimulation for their relief. The employment of morphine hypodermically has become an alarmingly frequent form of the opium habit, especially among members of the medical profession. Besides the general symptoms of chronic opium poisoning, we may have extensive ulcers and other local signs of skin irritation to deal with.

Ether and *chloroform*, habitually made use of, also cause serious disturbance of the nervous system; and so does *alcohol*. The abuse of spirituous liquors gives rise to a disorder of the mental, motor, and sensory functions, producing sleeplessness, headache, giddiness, hallucinations, imbecility, anæsthesia, disordered vision, and palsies. There results a fine irregular tremor, affecting particularly the hands, lips, and tongue, and occurring only on attempted movement. Multiple neuritis is also a common sequel. *Chronic alcoholism* also occasions a sensation of choking, a diminished vitality, a persistent catarrh of the gastro-intestinal membrane, a tendency to fatty degeneration, especially of the liver and kidneys; in short, the symptoms met with in drunkards and constituting the state described as chronic alcoholism. Chronic alcoholism in the parent may produce epilepsy in the child.

Chloral has proved, like opium and like chloroform, a very fascinating drug to many. The chief symptoms of chronic chloral poisoning are digestive disorders, irregular breathing, impairment of intelligence and of memory, persistent drowsiness, almost stupor, striking enfeeblement of will, want of power in the legs amounting at times to paralysis, and occasional tremor. Defective co-ordination with marked ataxic symptoms, similar to those of locomotor ataxia, and loss of knee-jerk, occur from the habit of taking chloral.[1] I have known delirium tremens to follow its use, when large quantities of it had been taken and the medicine stopped. Feeble, irregular action of the heart, and sweating, I have also found among the symptoms of

[1] J. C. Wilson, article "Opium Habit and Kindred Affections," System of Practical Medicine by American Authors, vol. v.

chloral poisoning. An erythematous inflammation of the skin of the fingers, with desquamation and ulceration around the borders of the nails, has been pointed out as a result;[1] and various forms of eruption, such as urticaria, lichen, and purpurous spots, as well as bedsores, have been observed after its prolonged use.

Paraldehyde is abused like chloral and morphine. It occasions, when taken habitually, gastric disorder, diarrhœa, sleeplessness, feeble circulation, sweating, and delirium tremens.

Tobacco used in excess gives rise to tremors, to giddiness, to emaciation, to impaired digestion, and to intermittence in the pulse, with irregular cardiac action and palpitations, which may become very annoying and originate the belief of an organic disease of the heart. Like the persistent abuse of alcoholic drinks, tobacco may occasion amaurosis; an insidious, obstinate form of otitis is developed in inveterate smokers, and is attended with very minute granulations of the pharynx, nasal fossæ, tubes, and middle ear.[2] When taken in large quantities by those previously unaccustomed to it, tobacco produces colic, diarrhœa, weakness, sleeplessness, dull hearing, vomiting, difficulty in breathing, cold sweats, feeble action of the heart, and will even cause collapse and death. The peculiar odor of tobacco may assist us in the diagnosis of tobacco poisoning: but it must be remembered that this may attend other morbid states in those who use tobacco largely.

Ergot, long continued, particularly when taken contained in impure flour, gives rise to the well-characterized disease, chronic ergotism. This appears mainly in two forms: the first is marked by convulsions with disturbance of sensation: the second by gangrene: both are apt to show themselves in epidemics. In the convulsive form there is at first formication, which lasts, whether attended with anæsthesia or not, throughout the whole illness. Soon muscular twitchings and cramps followed by painful contractions happen, and the convulsions may become general. These spasms especially affect the flexors of the arm, and unlike those of strychnine, they are not reflex spasms. There is no fever: the circulation is slow and feeble: the appetite is insatiable: we find nausea, vomiting, and diarrhœa. The disease generally lasts one or two months. In severe cases delirium occurs as a precursor to death. In gangrenous ergotism the same symptoms happen: but in addition we meet with gangrene without fever or signs of inflammation. The gangrene may be in the extremities or in the face.

[1] [illegible] [2] [illegible]

Let us now examine some of the features of slow poisoning by the metals.

Mercury, in any of its preparations, may lead to chronic poisoning. The mouth is inflamed, the gums are sore and swollen, the salivary glands act inordinately, and the breath is very offensive. Colicky pains, diarrhœa or bloody discharges, as well as acute nephritis, may occur. Tremors of the limbs when any motion is attempted are particularly frequent in cases where the poison has been inhaled in the form of vapor; they come on by degrees, and are associated with loss of power of locomotion. The tremors may be incessant and the movements involuntary, like those of chorea, and so rapid as to prevent the patient from obtaining rest at night.[1] In some cases an eczematous affection is observed. Poisoning by mercury is generally the result of the exposure to its action incidental to certain occupations, such as glass-plating, gilding, and working in quicksilver-mines; but it may be also noticed as following antiseptic injections of corrosive sublimate.

Lead poisoning is by no means uncommon among painters, plumbers, type-setters, and other workers in lead. Sometimes it may be caused by accidental circumstances, as when the patient has drunk water passed through leaden pipes, or taken snuff which has been impregnated with lead for the purpose of coloring it. Poisonous properties are also acquired by snuff wrapped in lead-foil; and lead poisoning has been observed after the use of cosmetics, and among those engaged in the manufacture of lucifer matches, of brushes, of lace, or working in glass enamel or glass powder;[2] and in consequence of food adulteration, especially of the use of lead chromate to color cakes.[3]

In such cases, the physician may have to depend entirely upon a correct appreciation of the symptoms for the diagnosis. Pain and uneasiness in the course of the colon, constipation, loss of appetite, anæmia, weakness, mental depression, and emaciation are the earlier signs. A metallic taste is perceived; the breath is fetid, the tongue pale and furred; the gums are edged with a narrow blue line of sulphide of lead, deposited mainly outside loops of blood-vessels. Colicky pains occur from time to time, and a severe and long-con-

[1] As in a case reported by Taylor, in which the patient died from the effects of the poison, without, however, having presented salivation or mercurial fetor of the breath, or a blue line on the gums. Guy's Hospital Reports, 3d Series, vol. x.

[2] Lacharrière, Arch. Gén. de Méd., Dec. 1859.

[3] Stewart, Clinical Analysis of Sixty-four Cases of Poisoning by Lead Chromate, Medical News, Dec. 31, 1887, and *ibid.*, Jan. 26, 1889.

tinued attack of colic may form the culmination of the disease. The muscles atrophy; electro-muscular contractility to the faradic current is greatly diminished, to the galvanic current it is frequently unaltered or increased; the sensibility of the skin is but little affected. Occasionally wrist-drop or paralysis of the extensor muscles of the forearms, the well-known phenomenon of lead poisoning, happens among the first symptoms; but it is more generally preceded by one or more attacks of colic. The right arm mostly suffers first. We also find at times lesions of the tendons in saturnine palsy. Yet a paralysis of the extensors occurs which is not due to lead, as in alcoholic multiple neuritis.

Another manifestation of lead poisoning is found in the severe pains in the joints and the neighboring muscles. These pains have violent exacerbations, and may be associated with cramps of the painful muscles. They are most common in the lower extremity, especially over and near the knee-joints. There are no signs of inflammation of the affected joints and muscles; pressure tends to relieve the pains.

Sometimes, in cases of saturnine poisoning, there is evidence of grave cerebral disorder: epileptiform convulsions, attacks resembling apoplexy, or general tremors and extended paralysis of the muscles, with acute delirium, inequality of the pupil, optic neuritis, retinal hemorrhages, loss of sight, and other signs of nervous disturbance, are noticed. Of course the diagnosis, under these circumstances, will be materially assisted by an accurate knowledge of the previous history of the patient as regards exposure to the action of the poison. The tremors are, like those caused by mercury, peculiar in ceasing when the limbs are supported or at rest; they are increased by movement. There may be tremor in the muscles of the face, which, however, are not affected by paralysis. Another result of lead poisoning is that it leads to granular degeneration of the kidneys. This is apt, again, to coexist with a gouty condition, which, as Garrod has shown, is one of the results of the absorption of lead. But the kidney affection may be found whether or not the joints are markedly affected, and may exist without albuminous urine.[1]

In instances in which the symptoms of lead poisoning are obscure or conflicting, we may search for lead in the urine. But the detection of small amounts of lead cannot be undertaken except by a professional chemist. A considerable proportion of the lead is eliminated by the bowels.

[1] Lancereaux, Arch. Gén. de Méd., Dec. 1881.

Copper-poisoning gives rise to dyspeptic symptoms, to diuresis, to loss of flesh, to lassitude and giddiness, to a peculiar greenish-blue perspiration, and to a green line on the gums and teeth. It is said that workmen in copper are singularly insusceptible to cholera or choleraic diarrhœa,[1] and that wounds in them heal with extraordinary rapidity. Copper appears to be somewhat less liable than mercury, lead, arsenic, or antimony to cause serious chronic poisoning, possibly because it is less cumulative. Small amounts of copper are frequently present in the liver and brain of man and some of the lower animals, also in some articles of food. Dr. Leffmann informs me that, in the examination of viscera from cases of lead poisoning which occurred in Philadelphia, copper in minute amounts was frequently encountered, and in one instance, that of a child four years of age, an appreciable quantity was obtained from a portion of the liver.

Arsenic, administered in small doses for a lengthened period, produces a state of chronic inflammation of the alimentary canal. Conjunctivitis, œdema of the face and the limbs, in some instances associated with albuminous urine, irritability of the stomach, diarrhœa, sleeplessness, increasing weakness, numbness, formication, alterations of sensation, and even paralysis, mark the progress of these cases; the hair and the nails occasionally fall out, and there is much frontal headache. Similar effects are noticed to follow the pernicious habit of arsenic-eating, and will be also encountered among persons employed in making artificial flowers and toys, in dyeing cloths, in manufacturing and hanging green wall-papers, or in the sublimation of arsenical ores; those, too, who live in rooms hung with papers containing much arsenic have exhibited the influences of the poison.[2] Besides the phenomena of internal. poisoning, cutaneous eruptions occur from arsenic. The extensors of the hands and feet are especially affected. In some instances, said to be not uncommon in Russia,[3] paralysis of the extremities, with muscular atrophy, happens. Arsenical paralysis may have mainly the symptoms of poliomyelitis, as I have had occasion to observe.[4] In other cases there are severe darting pains in the arms and legs, defective cutaneous sensibility, loss of knee-jerk, and the appearances of locomotor ataxia.[5] The palsies

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[1] Clapton, Clinical Society's Transactions, vol. iii.

[2] James Putnam, Analysis of Twenty-six Cases, Bost. Med. and Surg. Journ., March, 1889.

[3] Scolosuboff, Arch. de Phys., Sept. 1875.

[4] Phila. Med. Times, March and July, 1881.

[5] Dana, Brain, vol. ix.

of arsenical poisoning are now generally thought to be due to peripheral neuritis.

The inhalation of the fumes of *zinc* gives rise to a peculiar form of poisoning, characterized by a sense of weariness, by a feeling of tightness in the chest, and by attacks of shivering, followed by heat of skin and a profuse sweating-stage. This irregular form of ague is common among brass-founders.[1]

Carbon disulphide produces toxical effects of a singular character, conspicuous among which are gastric disturbances, inordinate appetite, loss of muscular strength, a cachectic condition, a feeling of icy coldness in the lower limbs, severe cramps in the calves of the legs, impotence, and, in severe cases, amaurosis, impaired hearing, hallucinations, loss of memory, and complete perversion of the intellect.[2] These phenomena are met with among workers in india-rubber.

Phosphorus is often seen, particularly among those who work in lucifer-match factories, to give rise to serious lesions. When the poisoning is caused by inhaling the vapor, it may occasion, as acute phosphorus poisoning does, alteration of the composition of the blood, a hemorrhagic diathesis, a fatty degeneration of several organs, as well as of the voluntary muscles,[3] and peptonuria. It also produces chronic bronchial catarrh, but especially necrosis of the jaw, for which the whole lower jaw has been removed.[4] The disease begins in carious teeth, and may extend to the cranial bones. Osteophytes form freely in the affected bones. Phosphorus taken internally in doses that gradually exert a poisonous effect leads to chronic inflammation and thickening of the stomach, colicky pains, diarrhœa, hectic fever, general emaciation, falling out of the hair, and to palsies, which are generally the precursors of a fatal termination.

Animal Poisons.—These may give rise to chronic as well as to acute poisoning. We find, for instance, syphilis, gonorrhœa, hydrophobia, dissecting wounds, snake-bites, acute glanders, and farcy,—all disorders exhibiting the effect of an animal virus. But we have already discussed some of these as far as is admissible in a work of this kind; and of the others it need only be said that the antecedent circumstances generally place the diagnosis beyond a doubt.

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[1] Greenhow, Med.-Chir. Transact., 1862.

[2] Delpech, Mémoires de l'Académie de Médecine, 1856; and Heurtaux, Recueil de la Société Médicale d'Observation, 1860.

[3] Lancereaux, L'Union Médicale, 1863.

[4] Cases of Hunt and Boker, Amer. Journ. Med. Sci., April, 1865; Wells, New York Med. Journ., Jan. 1866; Wegener, Virchow's Archiv, Bd. xl.

Yet there are a few illustrations of animal poisons and their effects which must be here, however briefly, mentioned.

One of these is the *malignant pustule* or *anthrax*, a terrible malady, which is the cause of many deaths on the Continent of Europe, and which is identical with the *charbon* of animals. The disorder is also prevalent in New Mexico.[1] It is communicated to man by direct inoculation; or by means of the skin or hair of the diseased beast, or by eating its flesh; or by insects which, sucking the poison from the sick animal, implant it in the skin of man. The poison produces a red speck, which develops into a vesicle, under and around which an extremely hard spot forms that becomes gangrenous. The surrounding skin inflames, new vesicles or pustules spring up, and the gangrene spreads rapidly, the patient speedily sinking; or the death of the parts is arrested, and separation takes place between the living and the gangrenous textures. In some cases it is attended with extended œdematous swelling and infiltration of the areolar tissue spreading from the anthrax pimple. It is remarkable how little local pain attends the grave constitutional disturbance, and the signs of low, irritative fever. The disease is found on the exposed portions of the body, as on the neck and hands. It has been traced by Davaine to the presence of filiform bacteria, bacillus anthracis. The blood swarms with these bacilli.

Closely connected with malignant pustule is the so-called "*wool-sorter's disease.*" The wool from sheep is not nearly so dangerous as the hair from the goat, the alpaca, and the camel. The symptoms may be those of malignant pustule with secondary splenic fever, or there often is an utter absence of either external or internal pustule.[2] The manifestations of the disease are a low fever with secondary abscesses, pyæmic symptoms, and pleuro-pneumonia. The complaint is a dangerous one; when ending in recovery, convalescence is slow.

Another disease transmitted from infected animals, and popularly known as the "lumpy jaw," is the so-called *actinomycosis hominis*, described chiefly by Israel[3] and by Ponfick.[4] The malady first appears in the lower part of the face, in the shape of little abscesses containing yellowish granules, which consist of ray fungi. These vegetations are readily detected by the microscope. The disease spreads to the ribs and vertebræ, and produces great destruc-

[1] A. H. Smith, Amer. Journ. Med. Sci., April, 1867.

[2] Bell, Lancet, June 12, 1880.

[3] Virchow's Archiv, Bde. lxxxv., lxxviii.

[4] Die Actinomykose des Menschen, Berlin, 1882.

tion of tissue: it is also found in the liver and the lungs, in the brain, in the intestines, and in the skin; there are the symptoms of chronic pyæmia. The affection may be mistaken for tubercle, stroma, or malignant tumor. Various forms of it, as of the liver and of the lungs, have been already described in connection with those organs.

The *foot and mouth disease* is an affection from which especially children suffer who have drunk the milk from infected cows. The poison produces an aphthous stomatitis with digestive disorder, and frequently also a vesicular eruption on the face and on the fingers and hands, which gradually dries into brownish scales, and at times a similar eruption between the toes. The disorder is not a serious one. It is due to a micro-organism, the streptocytus of Schottelius.

There is another form of animal poisoning which may be in this connection briefly considered,—namely, *milk-sickness*. It prevails in some of the new settlements of the southern and southwestern parts of the United States, and is brought on by drinking the milk or eating the flesh of cattle which have been exposed to certain influences the nature of which is unknown. Gastritis and enteritis are more or less blended in the early stage of the disorder, which at a later period resembles typhoid or typhus fever. The symptoms are lassitude, nausea and vomiting, with a sense of burning at the epigastrium, great oppression, intense thirst, fever, swollen tongue, obstinate constipation, fetid breath, and obvious abdominal pulsation. If at all, recovery takes place tardily, the tone of the stomach being often impaired for life.

There are forms of animal poisons originating in [illegible] during decay. The poisoning by these *ptomaines* from milk and eggs and other substances has already been mentioned. Frequently the ptomaine poisoning resembles that of the vegetable alkaloids, such as of morphine, codeine, and veratrine.

Besides these forms of poisoning, we find morbid states produced by animal poisons which arise from decomposing bodies or excretions, or from the crowding of many together, particularly of those of unclean habits, or of the wounded. These poisons reach the blood for the most part by the lungs, in the shape of *poisonous emanations*. They are very depressing in their action, may lead to low fevers, or to septicæmia, and in the case of the wounded to hospital gangrene. Persistent nausea, too, and a lowering of vital energy are not uncommonly observed in those who breathe continuously foul air under the circumstances alluded to,—as in hospitals, and in prisons in which cleanliness is not enforced and the regard is not paid to ventilation.

In some persons deleterious emanations from the human body give rise to a form of toxæmia, one of the chief features of which is the marked anorexia which attends the great debility.[1]

The exposure to animal effluvia may also excite violent diarrhœa, or even symptoms like those of cholera, certainly like those of severe attacks of cholera morbus. Of the occurrence of the former we have an illustration in the *dissecting-room diarrhœa*, which is usually attended with very fetid discharges, and may be accompanied by colicky pains, by nausea and vomiting, and by headache. The same kind of diarrhœa also happens in those who clean privies, or who are exposed to the emanations arising from sewers; or dysentery or choleraic attacks may follow the exposure. Nay, as in instances recorded by Becquerel, the instant disengagement of large quantities of putrid gases, arising from bodies far advanced in decomposition, where coffins have been opened, has caused sudden deaths, or has resulted in so serious a state of poisoning as to give rise to grave illnesses, having mostly a fatal termination.[2] In individuals who, in consequence of their vocation, are habitually brought in contact with animal effluvia and inhale noxious gases, besides the attacks of diarrhœa referred to, chronic disturbances of the stomach and liver, with marked impairment of the general health, may happen. Cases occur, too, of self-infection from ptomaines resulting from decomposition of fecal matter lodged in the cæcum, or by perforations taking place from the intestine into abscesses near by, into which the contents of the bowel find their way.

PARASITES.

Parasites are organisms which become secondarily implanted within or upon the body. Some parasites give rise to no symptoms at all; many occasion phenomena closely resembling those of other irritations. In any case, the only absolutely convincing evidence of the presence of a parasite is obtained by seeing it.

Vegetable Parasites.—The chief vegetable parasites have been mentioned in connection with diseases of the skin; the oïdium albicans, present in thrush, and the sarcinæ ventriculi, have also been described. All these vegetable growths can be detected only by the microscope; and, particularly in those involving the skin or the hair, it is of the utmost use to employ liquor potassæ, under the action of which the structures become transparent.

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[1] See Hunt's case, described by himself, in Pennsylvania Hospital Reports, vol. i.

[2] Traité d'Hygiène, 3d edit., p. 218.

Aspergillous infection of the lung, with hæmoptysis and cavity-formation, in persons engaged in carding hair obtained from rag-pickers, has been observed by Renon.[1]

A similar fungus that penetrates the internal tissues, the chionyphe Carteri, gives rise to that terrible disease known as *podelcoma*, or the *fungus foot* of India,—a complaint found among the natives of India who go about with naked feet. The fungus, introduced either through a scratch or passing through the pores of the skin, soon spreads, eating its way into the bones of the tarsus and metatarsus, and into the lower end of the tibia and fibula, producing a breaking up and absorption of the osseous tissues. The fungous particles or masses are generally of deep-black color, firm and globular, though they may be white or pinkish. The foot is enlarged about the ankle and over the instep; and on each side of the ankle-joint, and on the dorsum as well as on the sole of the foot, are small, soft swellings, having pouting openings that lead to fistulous canals communicating with the bones, which they perforate in every direction. The fungous mass is for the most part situated in the cavities in the bones, and from the canals passing to them transudes a discolored, glairy, or purulent and fetid fluid. The toes are distorted, and the muscles of the leg atrophied: but the fungus does not spread up the leg. The tendency of the disease is to cause death by exhaustion; the only remedy is amputation.[2] The affection has also been observed in this country.[3]

A similar disease, leading to local destruction, is the *perforating ulcer of the foot*. It is very uncommon in this country, although I have known of cases; in France it is not uncommon. It is supposed to be due to defective vitality of the parts from altered nerve-supply and the presence of pathogenic micro-organisms. Local anæsthesia, lowered temperature, and a tendency to profuse perspiration exist. The ulcer leads down to diseased bone. It is generally situated on the first or the last toe, over the articulation of the metatarsal bone with the phalanx.

The toes sometimes drop off from a disease which constricts them and enlarges them beyond the point of constriction. The affection is

[1] Monograph, Vienna, 1896, Comptes-Rendus de la Soc. de Biol., Nov. 1, 1895.

[2] See Carter, in Transact. Bombay Med. and Phys. Soc.; and on Mycetoma, or the Fungus Disease of India, London, 1874.

[3] Kemper, American Practitioner, Sept. 1876. Cases reported by Adami and Kirkpatrick in Montreal Medical Journal, Jan. 1896; by Hyde and Senn, Journal of Cutaneous and Genito-Urinary Diseases, Jan. 1896; by Pope and Lamb, New York Medical Journal, vol. lxiv., 1896; by Wright, Trans. of Apoc. of Amer. Phys., 1898; and by Arwine and Lamb, Amer. Journ

not unusual in Brazil, and seems to be peculiar to the negro. It is known as *ainhum*.[1]

Infectious, multiple gangrene of the skin may be caused by different varieties of micro-organisms. It has been found due to them in tuberculosis and in typhoid fever.

Animal Parasites.—When speaking of the affections of particular structures, some of these intruders have been mentioned,—those found in the skin or in the liver, for instance. It remains to consider chiefly such of the more important ones as inhabit the hollow viscera, certain solid organs, and the muscles.

Intestinal worms are the most common of all parasites. The general symptoms induced by them are those of intestinal irritation with disordered digestion. The appetite is capricious; the bowels are irregular, sometimes constipated, sometimes relaxed; the abdomen is frequently swollen and hard, and the seat of uneasiness or of colicky pains; the tongue is furred; the breath is fetid; and there is constant itching about the nostrils and anus. The patient, furthermore, grits his teeth during sleep, and is often annoyed by nightmare. Nervous disturbances are also met with; they may range from mere fretfulness to delirium, convulsions, chorea, epilepsy, or insanity. Strabismus and amaurosis may be also due to worms.[2]

There are many kinds of worms known to infest the alimentary canal of man, and they belong to the order of *nematoda*, or round worms, or to that of *cestoda*, or tape-worms.

The round worms are parasites of an attenuated or cylindrical form, and present these varieties:

1. The *ascaris lumbricoides*, or *round worm*, bears a considerable resemblance to the common earth-worm. It inhabits the small intestine, sometimes finding its way into the stomach, or even into the œsophagus, or being discharged through the abdominal parietes.[3] When it ascends to the stomach and œsophagus it causes sudden attacks of fever and gastric derangement, with nausea and vomiting; and even, at times, marked delirium.[4] The worms have been known to be so numerous as to obstruct the intestine.

2. The *oxyuris vermicularis*, *thread-worm* or *seat-worm*, is very small, the male being about two lines, the female about five lines in length. The parasite is white, slender, and extremely active; it is

[1] Da Silva Lima, Arch. of Dermatol., Oct. 1880; Duhring, Amer. Journ. Med. Sci., Jan. 1884.

[2] Hogg, Brit. Med. Journ., July, 1888.

[3] Garnier, L'Union Médicale, Oct. 1861.

[4] Schmidt's Jahrbücher, No. 10, 1868.

found in the anus, and causes intense itching of this part. The annoyance is sometimes such as to excite a suspicion of the existence of piles. It may creep into the vagina, giving rise there to profuse discharge; or into the urethra. It affects children frequently, but is not uncommon in adults.

3. The *ascaris mystax*, a parasite which inhabits the cat, may also infest the human body. It is a moderate-sized nematode, from two to three inches long, though the female may reach about four inches. Its head end is spear-shaped.

4. The *trichocephalus dispar*, or *long thread-worm*, is detected in very large numbers in the ileum near its termination, or in the colon, particularly at its head. It is from an inch and an half to two inches in length, and is characterized by the hair-like appearance of the head, which is generally buried in the mucous membrane of the intestine. It is not a common parasite, and it is doubtful whether its presence gives rise to any marked derangement. It has been found in the typh-fevers, and in persons dying from cholera or diarrhœa.

The *tape-worms* are jointed entozoa, of a ribbon-like form. They embrace the true tape-worms, or tæniadæ, and the bothriocephali. Of the former there are eight varieties, all of which have been found in man, though only two—the solium and the mediocanellata—are at all common; the tænia saginata, however, has spread over parts of Western Europe.[1] The bothriocephalus latus is the usual species of bothriocephalus met with in the human intestine; it, too, is increasing greatly in Europe, and, it is said, in Texas, particularly in the western portions.[2]

The *tænia solium*, or *pork tape-worm*, consists of an immense number of joints in connection with a single head. It inhabits chiefly the small intestines. The researches of Küchenmeister,[3] von Siebold,[4] and others have shown that its eggs become developed into the *cysticercus cellulosæ* discerned in the muscles of the pig, rabbit, and other animals whose flesh is used as food. Being once introduced into the alimentary canal, they find there a nidus in which to undergo development into the tape-worm. Cysticerci have also been detected in the muscles, the cellular tissue, the brain, the spinal cord, the heart, and the liver of man, and are most commonly met with in middle age

[1] Von Zehender, Parasitical Diseases of the Eye, Bowman Lectures, Deutsch. Med. Wochenschrift, No. 50, 1887.

[2] Colman, quoted in Sajous's Annual, vol. i., 1890.

[3] See Manual of Animal and Vegetable Parasites, Syd. Soc. transl., 1857.

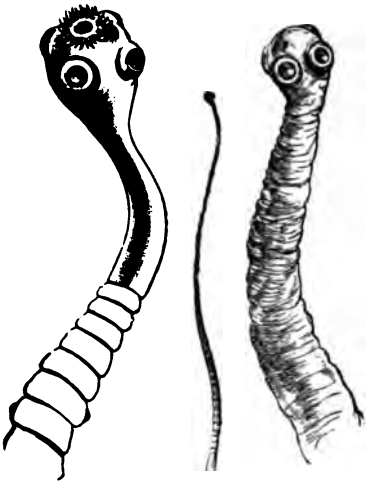
[4] Origin of Intestinal Worms, *ibid.*, 1857.

and in the destitute; they are the most frequent parasite in the eye.[1] They cannot, as a rule, be diagnosticated, except they be in positions in which they can be seen or felt, or the little tumors they occasion in the subcutaneous tissue be extirpated and examined. In the brain their chief symptom is violent and rapidly increasing epilepsy. In a case reported by Lloyd,[2] in which cysts developed in the right lateral ventricle and fourth ventricle, the symptoms were severe and constant headache, loss of memory, and a

FIG. 87.

Segments of tænia solium. Drawn from a specimen.

FIG. 88.



Heads of tæniæ, magnified, except the small central figure, which represents the head and neck of tænia solium, natural size. The figure to the left is the tænia solium, that to the right the mediocanellata.

sensation as if a ball were loose in the head and rolled about from the front to the back. There was left hemiparesis with ataxia, exaggerated knee-jerks, involuntary evacuations of the bowels, and failing vision, but no epileptic attacks.

The tape-worm is nourished from its head, the newly created flat segments pushing those already formed before them, so that the caudal

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[1] Bérenger-Féraud, Leçons de Clinique sur les Taenias de l'Homme, Paris, 1888.

[2] Transaction College of Physicians, vol. xx. p. 32, 1899.

extremity is the oldest portion of the animal. Each segment contains both a male and a female organ, the orifices of which are joined at the apex of a lateral papilla. In the *tænia solium*, the papillæ are arranged alternately at one side and the other. The size of the segments increases gradually towards the caudal extremity, the largest being three or four lines in breadth. There may be upward of eight hundred segments, and the worm may measure above thirty feet. Upon the head, which is about as large as that of a pin, is a double circle of hooks contained in sacks; the slender neck exhibits no segmentation. The sucking-disks in the *tænia mediocanellata* are larger than in the tænia solium, but the head, which is of blackish appearance and obtuse, has no hooks.

The tape-worm most frequently seen in this country is the tænia mediocanellata, or saginata, which is usually found in beef. Leidy stated, as the result of a large experience, that he had rarely encountered the pork tape-worm, *tænia solium*, as a parasite in the human intestines in this country. The habit of eating raw or partially cooked beef is the cause of much of the infection with tape-worm.

Tænia occasions disordered digestion, colic, cramps, a feeling of uneasiness in the abdomen, irritation of the mouth, nose, and anus, anæmia, headache, dizziness, disturbed sleep, mental depression, emaciation, cough, fainting-fits, cutaneous eruptions, and various cerebro-spinal affections, such as convulsions and epilepsy; yet there are no absolute data for the diagnosis of the parasite except the appearance of the links, segments, or proglottides in the discharges. In order that relief be permanent, the head must be expelled.

The *bothriocephalus latus*, *tænia lata*, or *broad tape-worm*, differs from the common tape-worm in having no lateral papillæ alternately arranged, but a single one at the centre of each segment; the segments themselves are much broader; the head is of elongated form, has no hooks upon it, and only a pair of fissures instead of the four mouths of the tænia solium, and we find no traces of joints until about three inches from the head. The parasite is of yellow or grayish-white color.

Roundworms, or lumbrici, belong also to the family of the nematoids. They may take up their abode in almost any organ in the body, especially in the liver, and are the immature brood of a species of nematoid, the adult form of the nematoid sometimes inhabiting the intestinal tract of boys. They consist at first of a vesicle having a corpuscle in its wall, a head upon which are six hooklets, variously arranged, but in adhering it is discarded. The embryo rises to hooklets, increases greatly in size, and becomes converted into a nem-

cle, around which a granular layer forms which afterwards becomes fibrous, constituting its capsule. The cysts develop in their interior a number of scolices, the larval form of the tænia echinococcus. The saline, non-albuminous fluid, contained in the tumor in large quantity, upon microscopical examination will usually show hooklets as well as scolices, thus absolutely establishing the diagnosis. A hydatid cyst may fail to develop any scolices, and is then termed an *acephalocyst*. The whole animal is surrounded by an investing membrane, which may burst and allow it to escape; the term hydatid designates the enveloping cyst and contents. When the echinococci are arrested in their normal development and are barren, not attaining to the production of scolices, they give rise to cysts with walls consisting of distinctly developed, concentric layers. When pressed tightly by the hand, they cause a peculiar gelatinous trembling or purring sensation.

The family of the *distomata* is not at all uncommon in man. A species of distoma, measuring from eight to fourteen lines in length, called the *distoma hepaticum*, usual in the liver and gall-bladder of the sheep, has been seen in the human liver and gall-duct, and also, it is said, in abscesses of the scalp. Other species of distoma have been found in the portal vein, ureters, kidneys, and bladder, and upon the intestinal mucous membrane; yet in the portal vein and its larger branches—a common seat of the distoma—the parasite produces little or no appreciable derangement; but when in the intestine it may give rise to congestion of the membrane, extravasation of blood, and the symptoms of dysentery. This has been specially noticed of the distoma hæmatobium, or Bilharzia hæmatobia, a worm common in Egypt, and the cause of the hæmaturia prevalent at the Cape of Good Hope and at the Mauritius. The entrance into the body is mainly through the urethra in persons bathing.

Filariæ have been met with in the blood and in the urine. The *Filaria sanguinis hominis*, according to Bancroft and Mason, gets into the system chiefly through the use of drinking-water in which the ova of this parasite have been deposited by mosquitoes, or by entering the skin of bathers. It gives rise to considerable pain in the loins, and leads to both bloody and chylous urine, and, according to Manson, to lymph-scrotum, the elephantiasis of the tropics. Thus far, I believe, only the filaria nocturna has been found in North America, and examinations for it must be made in the evening. Saussure[1] has reported twenty cases met with in Charleston, South Carolina. Mastin[2] proves that the filaria in the United States may be the cause of chylo-

[1] Medical News, June, 1890.

[2] Medical Record, Sept. 1888.

cele of the tunica vaginalis testis. Henry[1] reports a case of chyluria, appearing after a normal labor in a woman twenty-nine years of age, in which he discovered filaria nocturna in the blood. Dunn found active embryonic filaria in a case in which the symptoms were severe headache, fever, nausea, pain in the back, marked stomach pain and soreness, slight swelling of hands and feet, and puffiness of the face. The urine was suppressed for forty-eight hours, then chylous urine was passed, containing blood-cells, albumin, also leucocytes, oil-globules, and many embryonic filariæ.[2] Osler has placed on record a case of chyluria persisting for thirteen years in which no filaria was found. There is thought to be a non-parasitic as well as a parasitic chyluria.

A worm called the *strongylus gigas* has been observed in the kidneys. It produces hæmaturia, continuous pain, and an abdominal tumor,[3] and may lead to dropsy and death.[4]

The *dochmius duodenalis* is a worm producing a peculiar anæmia by sucking blood from the walls of the duodenum. It has been found especially among brickmakers, miners, and men working in tunnels, and the disorder has been identified by Leichenstern[5] with the so-called Egyptian chlorosis, tropical chlorosis, and brickmaker's anæmia. It has spread largely through Italian and Polish laborers employed in building tunnels, in mining, and in brickmaking. *Anchylostomiasis*, as the disease is called, is characterized by marked anæmia, by digestive disorder, abdominal pains, and bleeding from the bowels. There is a greater tendency to retinal hemorrhage than in simple anæmia.[6] Sandwith speaks of the marked sleepiness and dense stupidity.[7]

Fly parasites may be found in the dejections from the bowel and in the urine, producing local irritation of the intestine or the bladder.

The parasites which chiefly occupy the areolar tissues or the muscles remain to be described. Of these there are two of special importance.

One is the *filaria medinensis*, *dracunculus*, or *Guinea-worm*. This is a very slender, flat, finely ringed worm, which introduces itself into

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[1] Medical News, May 2, 1896, and Trans. Assoc. Amer. Phys., 1896.

[2] Transactions of the College of Physicians of Phila., vol. xx., March, 1898.

[3] Magner, Journ. de Méd. de Bordeaux, Feb. 1888.

[4] George, Med. and Surg. Reporter, Aug. 1888.

[5] Schmidt's Jahrbücher, Sept. 1888; also, Internationale klinische Rundschau, Oct. 1888.

[6] Discussion at the [illegible] Med. Journ., June, 1888.

[7] Trans. Elevent[illegible]94.

the subcutaneous areolar tissue: here it grows rapidly, and gives rise to swelling, with more or less inflammation; and sometimes to severe constitutional disturbance. After a time the swelling points and breaks, and the worm may be laid hold of and carefully twisted around a little piece of stick or a quill until it is extracted entire; if broken off, the eggs with which it is filled, getting into the wound, will become the agents of fresh mischief. Many of these worms may be found in the same patient, occasioning great annoyance and distress, even fatal exhaustion; but it is stated that there is often only one present. The number may vary between this and fifty. Some worms are twelve, others forty inches long, or even more. According to Busk, the parasite grows in the human areolar tissue at the rate of about an inch a week. Though it is most frequently found in the lower extremities, it has been observed to appear in the socket of the eye, in the mouth, the cheeks, the ears, and under the tongue and the scalp. It migrates rapidly from one part of the body to another. Where it exists, a pricking or an itching heat is felt; a vesicle forms when the worm is about coming to the surface, and this vesicle opens, leaving an angry-looking ulcer, in the centre of which the parasite shows itself. Phlegmonous spots may appear all over the body in which specimens of dracunculus are found.[1] The period of incubation is from eight to twelve months: a year often elapses before the Guinea-worm makes itself manifest in the human body.[2] The disorder, common in Asia and in Africa, is, fortunately, one with which we are unacquainted.

Trichina Spiralis.—This parasite was discovered by Owen in 1835 in human muscles taken from the dissecting-room; it was subsequently found by Leidy in the animal which it most infests, the pig; but it was not looked upon as other than harmless until in 1860 Zenker proved that trichinæ may exist free in the muscles of man, that they are encapsuled only after some time, and that they are the cause of a very serious disease.

The parasite is always introduced into the body by eating ham, pork, or sausages made from the flesh of pigs containing trichinæ. It is very probable that the hogs themselves obtain them from rats, in which they are common. It has also been stated that trichninæ may exist in beef; but this is not generally admitted.

The trinchina spiralis is the juvenile condition of a small nematode worm. It becomes fruitful only when introduced into the intestine.

[1] Woskresensky, quoted in Sajous's Annual, vol. i., 1889.

[2] Aitken's Practice of Medicine, vol. i.

After being swallowed, the female trichina begins to throw off minute embryos, which migrate to the muscular structures.[1] When the young trichina arrives in the muscles, it begins at once to destroy the muscular texture. It irritates the sarcolemma, leading to its gradual thickening and to an exudation that fixes the worm to a particular spot. Thus is formed the cyst which encapsules the parasite, and

FIG. 89.

Trichina in recent human muscle, taken the thirteenth day of illness. (After Dalton.)

which plays so important a part in its subsequent destruction. It takes a month or months for the cyst to form completely.

After the perfect formation of the cyst, further changes take place in it; particles of calcium and magnesium carbonate are deposited. The calcareous mass extends, and gradually covers the whole parasite, while around the prolongations of the cyst fat-cells are deposited. The whole process is very destructive to the flesh-worm, and it is thus that the disorder is cured. But it is apt to be months before this re-

[1] Leuckart, Untersuchungen über Trichina Spiralis, Leipsic, 1866.

sult is accomplished. Nay, as we know from two cases recorded by Virchow, neither the encapsuling nor the calcareous transformation kills the worms of necessity at all speedily; for in the one case they had remained alive for eight, in the other for thirteen and a half years after the infection, and in one instance mentioned by Turner[1] they were alive and active after twenty-six years.

The number of trichinæ in the muscles may be from several hundreds to many millions. Now, in accordance with their number in the muscles, with the character of the changes which there take

FIG. 90.

Trichina spiralis. Magnified 300 times. (After Virchow.)

place, and with the quantity in the intestines, will vary the extent of constitutional derangement and the signs of local irritation. Thus the symptoms and the dangers of *trichiniasis* are not always the same. When merely a few thousand trichinæ occupy the muscles, there are chiefly muscular pains with stiffness and general debility; signs which gradually ease as the worms become encapsuled and cretaceous alterations occur. When the muscles are occupied by millions of the flesh-worms, the local phenomena are much more severe; there may be almost complete immobility of the whole body, the muscles of respiration and of deglutition are implicated, irritative fever and general ca-

[1] Lancet, London, May, 1889.

chexia are marked, and the patient is apt to perish by gradual exhaustion, or in consequence of the disordered respiratory function, or of some pulmonary complication. The presence of large numbers of trichinæ in the intestine produces diarrhœa, vomiting, abdominal pain and tenderness; or the worms may shortly after being swallowed give rise to a kind of cholera morbus. Should the signs of the affection not appear until from twenty-one to twenty-five days after the use of the infected meat, and take the form similar to acute rheumatism of the joints, there are not as many trichinæ present as in the choleroid or the typhoid variety of the malady, each of which Rupprecht[1] has told us shows from five to ten millions.

FIG. 91.

Trichina capsule with shell-like calcareous deposits. (After Leuckart.)

Speaking generally, we may recognize in trichiniasis three stages: the first, lasting about a week, during which the trichinæ are being generated in the intestines and in which we find only signs of gastro-intestinal irritation; the second, the passage of the brood into the muscular textures, and the disturbances it there occasions; the third, the retrogressive formation, which fairly sets in about three or four weeks after the beginning of the second. Now, it is the last two stages which yield the most striking manifestations of the malady: loss of appetite; pasty taste in the mouth; nausea or vomiting; dry, somewhat coated tongue; diarrhœa; abdominal pain and meteorism; prostration; fever, with a quick pulse and copious sweating; œdematous swelling of the face, followed in grave cases by almost general anasarca; sensitiveness of the skin and the muscles to the touch, or painfulness when the latter are moved, and their contraction and difficult motion; dyspnœa; sleepless nights; nocturnal attacks of abdominal neuralgia; and emaciation. There is also decided leucocytosis.

FIG. 92.

Encapsuled chalky concretions in muscle, due to dead trichinæ. Magnified about thirty times. (After Leuckart.)

The *fever* is a marked symptom. It sets in early, owing to the

[1] Vierteljahrsschrift für Ges. Med., Oct. 1880.

intestinal irritation, though it is not until the end of, or after, the first week, after therefore the migration of the young trichinæ has fairly begun, that it is strikingly developed. The temperature is about 101°, though it may pass to 104° and 105°; yet it does not, as a rule, reach the high heat which is observable in other continuous fevers. But it is especially in the attending profuse perspirations, the absence of enlargement of the spleen and of an eruption, the swelling of the face, the muscular symptoms, and in a very red color of the visible mucous membranes, that the points of difference lie between the febrile excitement of trichiniasis and *typhoid fever*,—a malady which, on account of the continuous fever, the prostration, the diarrhœa, and the sudamina, it resembles. In light cases of trichiniasis there may be no fever, or there may be a fever more of intermittent or remittent character. The appearance of the face may be like that of *typhus* fever; here, however, the muscular pains are wanting.[1]

The *œdema* marks the beginning of the second stage of the affection. It manifests itself first in the eyelids, about the seventh day of the disease, and is attended with a catarrhal state of the conjunctiva, with dilated pupils, great susceptibility to light, diminished power of accommodation, and pain in moving the eye. The swelling may extend over the whole face, and is sometimes associated with flushing. It is uninfluenced either by the sweats or by the diarrhœa, but lessens generally very much, or even disappears, after lasting eight or nine days. But instead of the œdema subsiding, it may extend to the chin, to the arms and legs, and to the back. It is probably due to pressure upon the arteries, exerted by the parasites and the exudation. The dropsical swelling of trichiniasis is not associated with albumin in the urine. Still, we find occasionally a slight amount of albumin, as well as polyuria, though generally the quantity of urine is diminished. The trichinæ may at times be detected in the passages from the bowels.

The *muscular symptoms* begin in the second stage, at about the tenth day, with pain and stiffness in the limbs. The muscles are extremely painful when touched or moved; and the patient lies in consequence as quietly as possible. The immobility is also due partially to the retracted state of the muscles which occurs, manifest for instance in the semiflexed position of the extremities, and in the rigid, trismus-like setting of the jaws. The disturbance of function of certain muscles becomes particularly evident. The disorder of the

[1] See Clinical Lectures on Acute Trichiniasis, by J. M. Da Costa, reported in Medical News and Abstract, March, 1881.

muscles of the eye has been already spoken of; we encounter, besides, impaired hearing, difficulty of deglutition, and loss of voice, from the muscles of the ear, of the pharynx, and of the larynx being filled with trichinæ. The respiratory muscles are commonly much affected, and we find hurried and shallow breathing. The muscles of the heart usually, and the unstriped muscles of organic life constantly, escape infection; and, as the trichinæ wander to the front of the body rather than to the back, the muscles anteriorly are more infested than those posteriorly. An interesting observation, which may lead us to suspect the true cause of the muscular pains, is the great increase of the eosinophiles in the blood, to which Brown[1] has called attention. In a case mentioned by Osler[2] they reached sixty-eight per cent. of the total number of leucocytes. Large numbers of eosinophilic cells may be found in the muscles without there being an increase of these cells in the blood.[3]

The marked muscular pain, the stiffness, the fever, the profuse sweats, the acid urine, simulate the signs of *acute rheumatism;* but we find in trichiniasis diarrhœa, no articular swelling, and no heart-complications. Error is more likely to happen with reference to acute muscular rheumatism. But the signs of prostration and of gastro-intestinal irritation are here wholly wanting.

The condition of the respiratory muscles gives rise, as already stated, to the embarrassed respiration, but it is not the only cause of the *pulmonary symptoms.* Congestion of the lung, bronchitis, and pleuritis are usual. They are not uncommonly combined with pneumonia, which appears suddenly, selects the lower portion of the left lung by preference, occurs about the twenty-sixth day of the disease, and generally proves fatal. The sputa consist of dark, unmixed blood; and the pneumonia is thought to be due to a trichinous embolism, the clots being derived from thrombi, which form in the venous system.[4] Limited catarrhal pneumonia may be also met with.

If the patient escape a serious pulmonary complication, if he have strength enough to withstand the weeks of irritative fever and exhaustion, he enters at the end of a month or of five or six weeks of suffering upon a gradual convalescence. His appetite becomes insatiable, and he moves his limbs with more and more freedom. But it is a long time before he regains his full muscular power. Indeed, this

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[1] Johns Hopkins Hospital Bulletin, April, 1897, and Medical News, Jan. 1899.

[2] Practice of Medicine, 3d edit.

[3] Howard, Phila. Med. Journal, Dec. 2, 1899.

[4] Rupprecht, Trichinen-Krankheit, 1864.

may be always somewhat impaired. In some cases convalescence is greatly retarded by boils, by inflammation of the lymphatic glands, and by dropsy. Children convalesce more quickly than adults. They suffer, in truth, less from the disease, and are not very subject to it.

The diagnosis of the malady has been made evident while discussing the symptoms. At first the signs of gastro-intestinal catarrh, the vomiting, the slight fever, the perspiration, the muscular feebleness, are the most significant, and these early manifestations might be mistaken for *irritant poisoning;* we can tell their meaning prior to the marked development of the phenomena in the muscles only by the detection of trichinæ in the stools. The same may be said of *cholera morbus.* Again, it must be borne in mind that in some cases of trichiniasis the first symptoms of the complaint do not happen for two or three weeks after the infected meat has been eaten; and that in others it runs a chronic course and the whole disease is very protracted. The so-called "*sausage poisoning,*" not dependent on trichinæ, differs from trichiniasis in its rapid course and in the quick appearance of the choleraic symptoms after the spoiled sausages have been partaken of. In *periarteritis nodosa* the severe muscular pains are associated with thickening of the vessels, muscular atrophy, palsies, and great disproportion between the rapidity of the pulse and the temperature,[1] and an examination of the muscles will show the absence of the trichinous affection. Indeed, in any instance, no matter what be the complaint trichiniasis may simulate, there is, though we may suspect it from the eosinophilia, but one means of determining the presence of the flesh-worms positively,—to examine a piece of muscle. This may be effected by cutting down upon a muscle and removing sufficient of its structure for a microscopical examination, or by using Middeldorpff's harpoon or Duchenne's or Hart's trocar.

Owing to the œdema, and particularly the œdema of the eyelids and face, the malady may be confounded with *Bright's disease.* But the absence of albumin and tube-casts in the urine distinguishes it. The physical signs separate the dyspnœa it occasions from that of *cardiac disease;* and the sweats and the muscular symptoms of trichiniasis tell us what we are dealing with.

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[1] Schrötter, Wien. Med. Wochenschr., No. 15, 1899.

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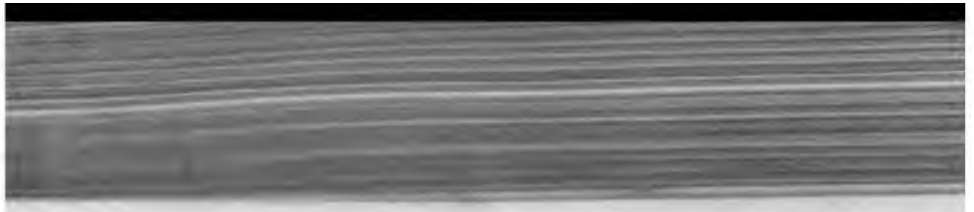

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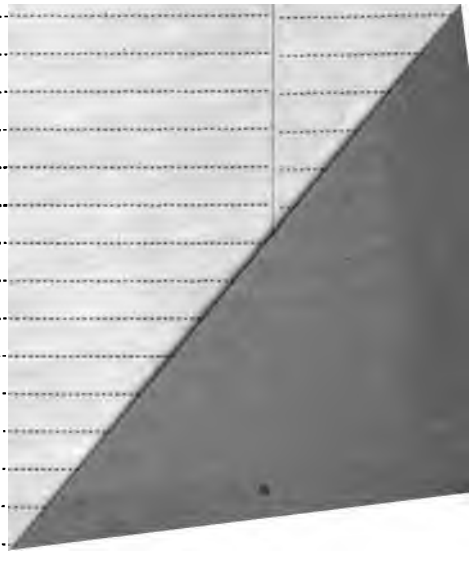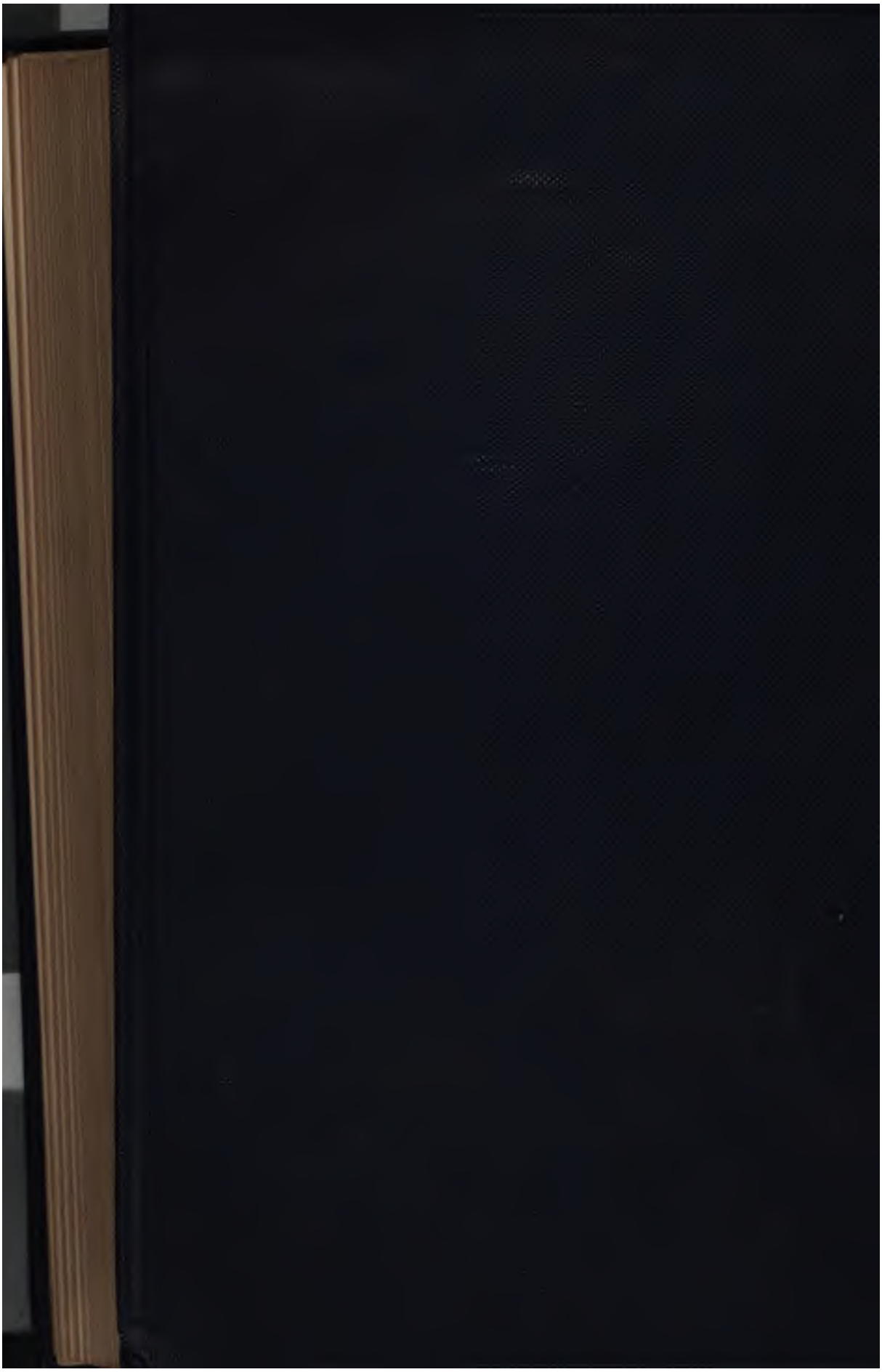
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